MAY, 1950

# The Review of Gastroenterology

OFFICIAL PUBLICATION

NATIONAL GASTROENTEROLOGICAL ASSOCIATION

Vagotomy: An Experimental and Clinical Appraisal of the Effect of Section of the Vagus Nerves at Different Levels

The Problem of Gallbladder Disease

Surgical Repair of Femoral Hernia

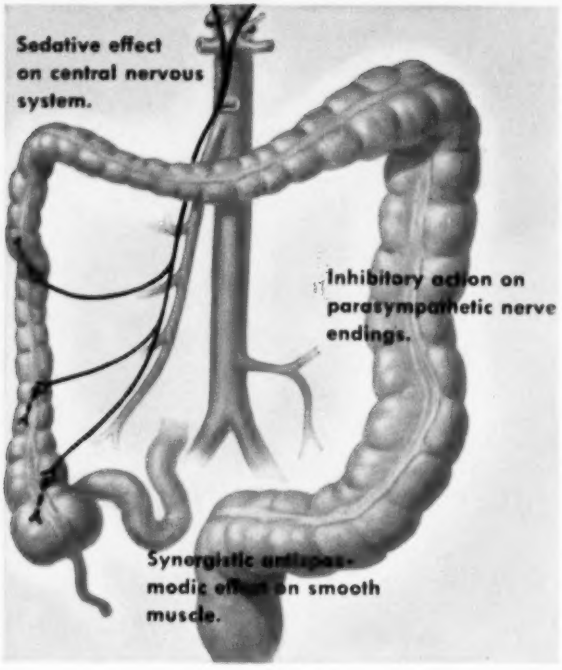
Experientia Docet

●

*Fifteenth Annual Convention*

*New York, N. Y., 9, 10, 11 October 1950*

VOLUME 17 NUMBER 5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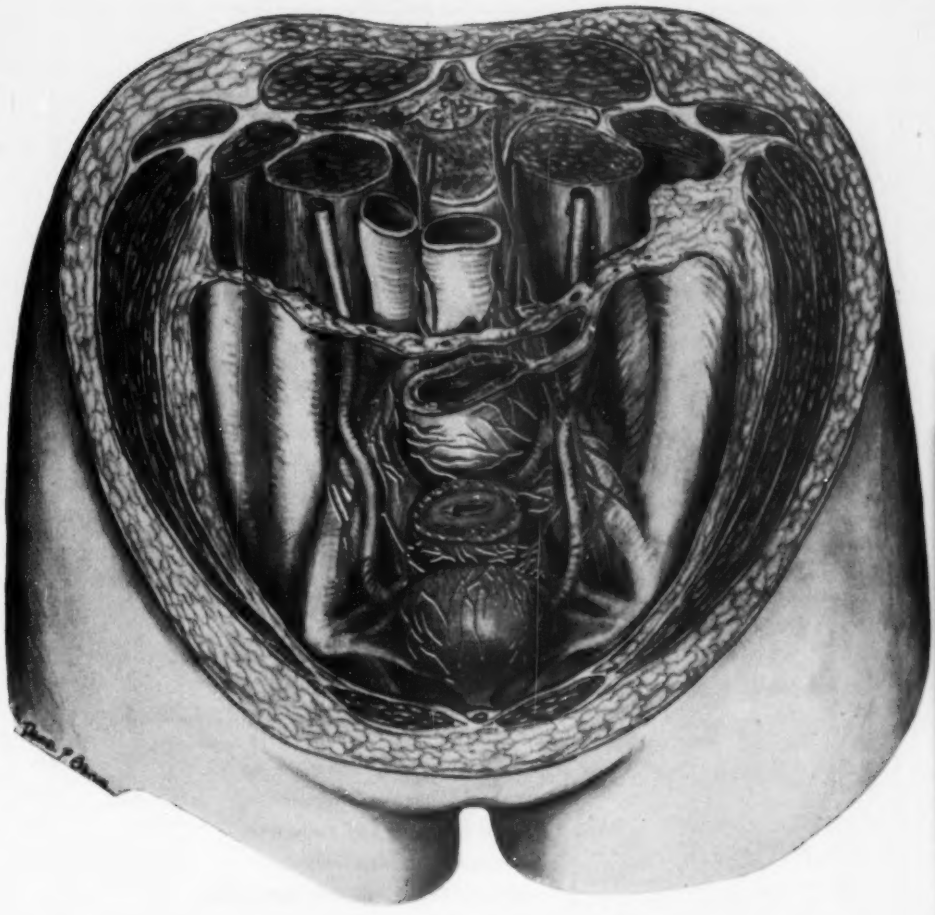

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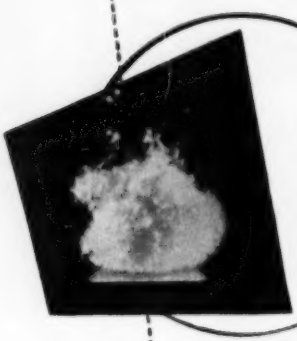

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*The Pioneer Journal of Gastroenterology, Proctology and Allied Subjects in the United States and Canada*

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2. Batterman, R. C., and Ehrenfe'd, L.: Gastroenterology, 9:141, Aug., 1947.
3. Woldman, E. E., and Polan, C. G.: Am. Jour. Med. Sc., 198:155, Aug., 1939.

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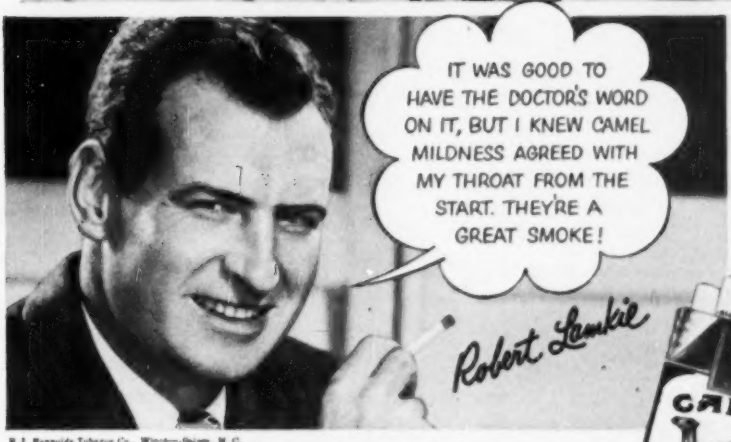
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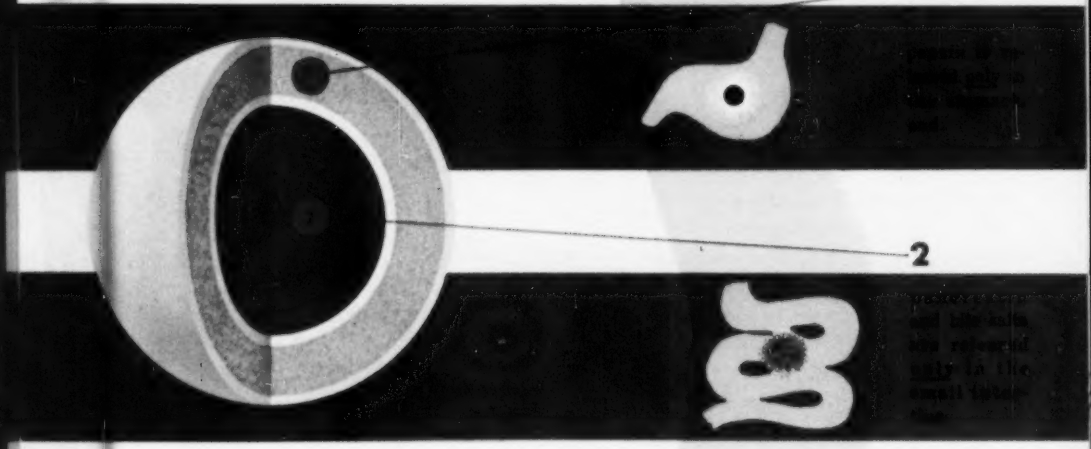


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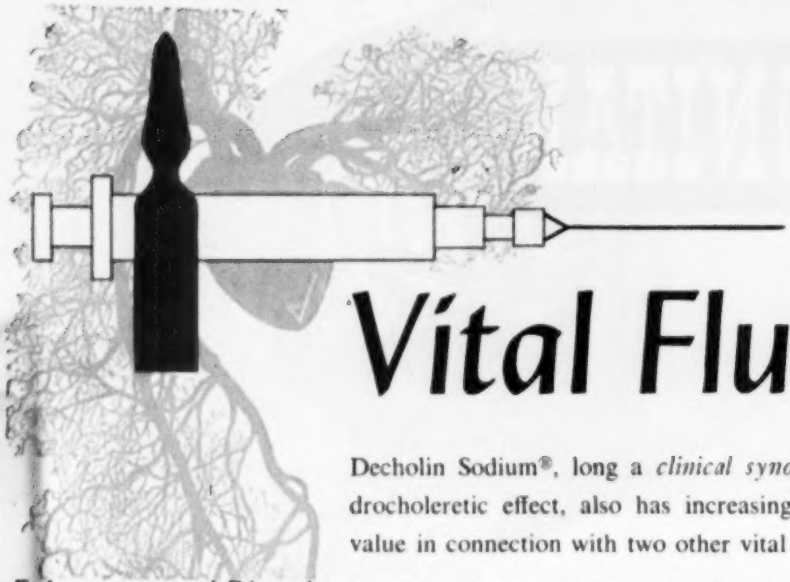

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Volume 17 MAY, 1950 Number 5

# THE PROBLEM OF GALLBLADDER DISEASE*

ALBERT F. R. ANDRESEN, M.D.
Brooklyn, N. Y.

That the treatment of disease of the biliary tract is a serious problem today is evidenced by the controversial nature of the literature on this subject; by the generally poor results seen in patients who have been treated either medically or surgically; by the many gallbladder patients who are being treated as difficult psychiatric cases, and by the increasing literature on the "postcholecystectomy syndrome". The term "gallbladder disease" or "biliary tract disease" usually includes not only cholecystitis, acute and chronic, but also cholesterosis, cholelithiasis, cholangitis, neoplasms, so-called functional disorders and allergic reactions. While these conditions do not all have the same indications for treatment it is important to understand the physiology of the biliary tract in order to get a clear conception of the etiology of its diseases and their proper treatment.

## Physiology

The functions of the biliary tract are quite clearly understood. Clear limpid bile, in quantity between 500 and 1,000 cc. in twenty-four hours is continually secreted by the liver and runs down through the hepatic ducts and common duct to the sphincter of Oddi. When this sphincter is closed, as it is when the duodenum is empty, the bile, not being able to enter the duodenum, backs up, and due to the fact that the secretory pressure from the liver is always greater than the pressure in the gallbladder, it is forced through the cystic duct into the gallbladder. In the gallbladder it is concentrated to from four to ten times its original density, thus enabling this organ to store large quantities of bile and providing a concentrated bile for aid in the digestion of fats when required. When food enters the duodenum, it results in the relaxation of the sphincter and the production of the hormone, cholecystokinin, which causes contraction of the gallbladder. Even with forceful contraction the gallbladder pressure does not exceed the hepatic secretory pressure, so that the concentrated bile, mixed with fresh bile coming down from the liver, is forced into the duodenum. While any food entering the duodenum from the stomach has been shown to stimulate the production of cholecystokinin, it has been demonstrated that fats, especially in the form of cream and egg-yolk are most efficient in this respect. Peptone is less efficient. Roentgenologists have made use of this knowledge by using these fats to test gallbladder function.

*Read before the Fourteenth Annual Convention of the National Gastroenterological Association, Boston, Mass., 24, 25, 26 October 1949.

It has been shown that the concentration of the bile increases with the length of time it remains in the gallbladder, so that at times it is so thick that its expression from the gallbladder becomes difficult or impossible. Increasing concentration and decreased frequency of evacuation of even a part of the contents tends to the formation of masses of cholesterin crystals, or of calcium bilirubinate, resulting in the formation of calculi. Chemical imbalance, especially disturbance in the bile acid-cholesterol ratio, undoubtedly also plays a part, but this is also the result of stasis. Infection also has been found more frequently in the presence of stasis. While it has been postulated that the degree of concentration of cholesterol in the blood may have something to do with the gallstone formation and that therefore the ingestion of fats should be avoided in the prophylaxis and treatment of biliary tract disease, it is easy to see the fallacy of this reasoning. In the blood the cholesterol content normally varies from 0.13 to 0.19 per cent, and in the presence of gallstones it has been shown to vary from 0.14 to 0.3 per cent. In fresh hepatic bile it varies from 0.45 to 0.6 per cent which becomes concentrated four to ten times or more in the gallbladder and constitutes 85 to 90 per cent of cholesterin stones. How can a concentration of cholesterol in the blood which is only one-quarter of its concentration in normal bile, produce an increased concentration in the gallbladder? No definite connection between hypercholesterolemia and gallstones has ever been proved. Besides this it has been demonstrated that ingested lipoids have no effect on cholesterol concentration in the blood nor upon the fatty acid-cholesterol ratio, so that the lipoid-free diet could have no effect in any event.

I am constantly seeing patients who have been on fat-free diets and consult me because their symptoms have not been relieved or because they are no better or are even worse than they were before operation. Clinicians who prescribe such diets seem not to realize the importance of fats aside from their stimulation of gallbladder emptying. Fats not only are the carriers of the fat soluble vitamins but have been shown to be important in the utilization by the body of the B vitamins and of calcium and phosphorus, and also of nitrogen and carbohydrates. Patients who have been deprived of these advantages become weak, emaciated and anemic and often show other definite evidences of dietary deficiencies. So-called dyskinesia or dyssynergia, or spasm of the sphincter of Oddi, usually attributed to neuropsychologic or reflex causes, is usually caused by insufficiency and infrequency of feedings containing fats and is of course aggravated by a fat-free diet. Adhesions between the gallbladder and neighboring organs may result from acute cholecystitis with or without perforation, from chronic cholecystitis or from infection or perforation in contiguous organs.

## Etiology

The etiology of cholecystitis is not clearly understood. While *acute cholecystitis* may occur in connection with an impacted stone or obstruction due to a neoplasm and may be due to direct infection or secondarily to generalized or focal infections, some deny that infection plays a part, as evidenced by frequent failure to find organisms in the acutely inflamed gallbladder, even in the presence of perforation.

These claim that "metabolic disturbances" are the cause. Allergy also at times may be the cause of acute gallbladder disturbances.

*Chronic cholecystitis* is another bone of contention. Some claim that there is no such condition and that what is usually called chronic cholecystitis is really only a functional disturbance. However, the chronic changes in the gallbladder wall seen even in the absence of lithiasis resemble those produced by chronic inflammation. Such chronic changes may result in shrinkage and even obliteration of the gallbladder and may result in neoplastic changes, benign or malignant, the latter fortunately with great rarity. Cholesterosis, or "strawberry gallbladder" is due to deposition of cholesterol esters and other lipoid materials in the wall of the gallbladder. The mechanism is probably similar to that producing gallstones and it is often associated with the presence of stones.

*Cholelithiasis*, at one time held to be due to infection, to the enclosing of bacteria in a shell of hard material, now is considered to be due mainly to biliary stasis, according to the mechanism described earlier in this paper, with infection, if present, a secondary matter. Any interference with normal biliary drainage would therefore be an etiological factor. Infrequent ingestion of food, especially of fats, is an important cause and accounts for the frequent development of gallstones during pregnancy, especially where morning vomiting has resulted in an insufficient frequency of feedings, particularly of fats. It also explains the gallstones seen in obese women who have been on very restricted diets for reducing weight, and in persons who pride themselves on eating only one or two meals per day and no between meal feedings.

The occurrence of *biliary colic* can usually be explained on a physiologic basis. It has been shown that colic is not due merely to the presence of stones, as only a small proportion of patients with stone-filled gallbladders experience attacks of colic. Even stones in the ducts often produce no pain. Lewis Gregory Cole demonstrated that pain occurred during the passage of a stone through the outlet of the gallbladder. Other experiments have shown that sudden distention of the gallbladder, with spasm at its outlet, will occasion pain. A gallbladder containing stones, becoming distended with bile as a result of insufficient and infrequent feedings, especially of fat, will suddenly contract following a large fatty meal and may force a stone into its temporarily stretched outlet, producing an attack of colic. A dietary history of this kind can be obtained in a large proportion of cases. Fasting during and after an attack of colic also accounts for the frequent occurrence of repeated attacks and points the way to prevention.

In acute or chronic inflammation and thickening of the gallbladder wall there may be interference with the entrance of bile into the gallbladder or, if it has entered, a diminution or failure of concentration and poor or absent gallbladder contraction. All of these phenomena are demonstrable by cholecystography. In such conditions or during an attack of biliary colic the passage of cream, oil, egg yolk or peptones through the duodenum will produce sphincter relaxation and permit bile to flow into the duodenum as a result of hepatic secretory pressure alone even though the gallbladder is not functioning. The conception that an

acutely inflamed gallbladder could be made to contract so violently that it could perforate is fantastic and is not in agreement with the results I have seen in feeding such cases early and often. I can see no contraindication to promoting biliary drainage by such feedings and this biliary drainage is desirable for the purpose of reducing pressure in the biliary tract under any conditions except the presence of an "acute abdomen".

## Diagnosis

Although at times the diagnosis is easy and can be predicated upon the old adage of "fair, fat and forty with indigestion", it is imperative that a complete and careful study be conducted in all cases. I see too many patients who have been treated for and even operated upon for gallbladder disease who either did not have it or who had some other, more serious condition elsewhere which was overlooked.

The *history* is important. Although symptoms may be entirely absent in a large proportion of cases even in the presence of gallstones, when present the symptoms are definitely attributable to disturbed physiology of the biliary tract and its neighboring organs or to mechanical difficulties. It must be remembered that while biliary tract pains are usually located on the right side below or above the rib margin, in the right lower quadrant, epigastrium or right subscapular region, they may at times be reflected to the left side and the only pains may be in the left upper quadrant or left chest. A chronic diseased gallbladder may be irritable and show spasm or increased contractions, especially if thick bile or calculi are interfering with proper emptying, producing biliary colic of greater or less severity, wholly or partially relieved by the expulsion of the plug. Or the gallbladder may be atonic, with resultant distention causing a feeling of fullness or pressure in the right upper quadrant or epigastrium. Although after a stone has been expelled from the gallbladder into the cystic duct it rarely causes pain, occasionally partial cystic duct obstruction may cause sufficient stasis to provoke colicky pains. When the stone reaches the wider common duct it causes no symptoms unless it becomes lodged at the outlet of the duct, when it may cause an increasing jaundice if it blocks the duct entirely or intermittent jaundice if it moves up and down (the "ball-valve stone"). If it is not finally expelled into the duodenum, the resulting icterus, the backing up of bile into the gallbladder, with perhaps development of acute cholecystitis or empyema of the gallbladder, and biliary cirrhosis may result in death unless the stone is removed by surgery or unless it perforates through the duct, in which case peritonitis may result or adhesions may cause occlusion or other complications. The perforation of a stone through the wall of the gallbladder or ducts is probably due more to necrosis from decubitus than to intraluminal tension.

Frequently the only symptoms are those due to pressure upon or irritation of neighboring organs. The pyloric region and duodenum respond to this irritation by causing *retrostaltic symptoms*, such as peristaltic unrest, fullness, distention, nausea, heartburn, sour eructations, regurgitation and vomiting, all or some of which may be constantly present or may occur in attacks of varying duration.

At times *diarrhea* may be the only symptom, explainable either upon irritation of the contiguous hepatic flexure or upon the achylia which often accompanies gallbladder disease of long duration, usually due to gastritis. Patients with retrostaltic symptoms frequently complain of "gas", swallow much air and belch excessively. Regardless of the cause of these symptoms, a dislike for food often accompanies them. A particular dislike for fats and oils may be purely psychological or may be due to their "feel" in the mouth. In gallbladder disease, occasionally some distress also may result from the fact that the fats stimulate activity in the diseased organs. It must be borne in mind that these retrostaltic symptoms occur in many other conditions than gallbladder disease. They are common in peptic ulcer, gastroduodenitis, any other gastrointestinal lesions and in pelvic, renal or even pulmonary and cardiac disease. At times the pains of gallbladder disease may be simulated by those due to the lesions just mentioned or to skeletal, spinal cord, neuromuscular or arthritic conditions. Frequently symptomless gallbladder disease is accompanied by pains due to spinal arthritis, angina pectoris, renal calculus or other conditions. In such cases the gallbladder is often removed because calculi have been found and symptoms of course persist. Another common error of diagnosis results from the interpretation of a jaundice, with or without pains, as being due to gallstones when careful study would disclose the cause as being a blood dyscrasia, hepatitis, an advanced cirrhosis or even carcinoma of the liver.

*Acute cholecystitis* produces symptoms and signs which usually are not possible to differentiate from any other cause of an "acute abdomen". Even a previous history and definite findings of gallbladder disease or cholelithiasis will not be of help in the diagnosis. It must be remembered that acute cholecystitis may develop as a result of common duct obstruction from an impacted stone or from a neoplasm and will then be accompanied by jaundice. Acute pancreatitis is often a result of the backing up of bile into the pancreatic duct because of blockage of the common duct by gallstones, and perforated peptic ulcer is not an uncommon complication. Acute appendicitis, Meckel's diverticulitis, hepatic or subphrenic abscess, pneumonia, pleurisy, coronary thrombosis (which may co-exist), mesenteric thrombosis, intestinal obstruction, renal colic, lead colic or an allergic reaction may also simulate acute cholecystitis.

*Examination* of a patient with gallbladder disease should be complete. Search for focal infections in mouth, nose, throat and pelvis should be thorough. Rectal, digital and proctoscopic examinations should be made and esophagoscopy and gastroscopy may be indicated. The pulmonary and cardiovascular-renal status should be definitely evaluated. Neuropsychiatric factors should be considered. Abdominal examination in acute cholecystitis will show the usual signs of an acute abdomen, but chronic gallbladder disease may present no abnormal abdominal findings except the frequently accompanying obesity. Tenderness, spasticity or the feeling of a mass resembling the gallbladder may be of help in diagnosis, but too often these findings will occur when the biliary tract is entirely normal, so

that unless acute symptoms demand immediate care, treatment should not be instituted until an adequate laboratory study has been completed.

*Laboratory examinations*: Blood, urine and stool examinations, cultures, hepatic, pancreatic and renal function tests, biopsies, paracentesis and electrocardiographic studies may be helpful or absolutely necessary to make a differential diagnosis, to determine the severity of the disease, to rule out complications and to evaluate the general condition of the patient as regards operability.

*Gastric analysis*, by the fractional method, using histamine as the stimulant to gastric secretion, is of value not only in determining the degree and amount of gastric secretion and motility, but may disclose bleeding. Blood in the gastric content may indicate ulceration or malignancy in the esophagus or the stomach, blood found only in regurgitated duodenal contents may be due to duodenal ulcer, to the trauma of a recently passed gallstone or to some other cause for bleeding within the biliary tract.

*Duodenal biliary drainage*, not used as much now as formerly, may show large numbers of cholesterin or calcium bilirubinate crystals, indicating that calculi may be forming, may show the bleeding just mentioned, occasioning a suspicion of malignancy or may show an absence or diminution of biliary or pancreatic secretion or both, indicating the degree of a common duct obstruction or of pancreatic dysfunction.

*Roentgenologic study* is most important. Even in the presence of an "acute abdominal calamity" scout films may show the presence of opaque calculi or may disclose evidence of perforation of a hollow viscus. In the presence of chronic symptoms suggesting gallbladder disease it is unfortunately a frequent custom to base the diagnosis only upon a cholecystographic study. Neglect of a complete and thorough gastrointestinal study may have tragic consequences. I frequently see patients, whose cholecystograms indicated gallbladder disease, who have been treated or operated upon for this and who are shown afterwards to be suffering from a gastrointestinal malignancy which was overlooked and permitted to advance to an inoperable stage. A *complete gastrointestinal series*, with careful fluoroscopic and radiographic evaluations, including esophageal and barium enema studies, should be done in every case, not only to rule out other disease but to observe pressure effects or adhesions from the biliary tract and pancreas and their effect on gastrointestinal motility. The frequent association of duodenal ulcer, chronic pancreatitis and appendicitis with gallbladder disease must not be forgotten.

*Cholecystography* has become pretty well standardized as to technic, but interpretation is still a moot point. With modern rapid technic in most cases not only the dye-filled gallbladder but also the cystic duct and often the common duct can be well seen, at least in some of the films. Dilatation, deformity and partial obstruction of the ducts can be demonstrated. It should always be preceded by one or more plain scout films, looking for opaque calculi, a thickened or calcified gallbladder and the presence of other tumors or calcified areas. The diagnosis of liver enlargement by roentgen observation is often fallacious and

must be carefully checked by physical examination. At the outset it must be emphasized that in order to visualize the biliary tract the dye-stained bile must be flowing freely through the tract. It is also a fact that the dye is a toxic substance which the liver excretes through the bile. Therefore cholecystographic study is contraindicated in the presence of a badly damaged liver, in acute biliary tract disease and in jaundice from any cause. The shadow of the dye-filled gallbladder and ducts should be studied at different angles, including films in the erect posture, showing the degree of motility, the presence of fixed deformities or of fixed defects or rarefied areas caused by neoplasms. If the gallbladder is well filled with the dye, careful study of its density and homogeneity will usually determine the presence or absence of calculi, which can be confirmed later by films taken after the fatty meal, when, with less free dye in the lumen, the calculi, often impregnated with the dye, will stand out more prominently. Until the type of fatty meal and the time and frequency of postprandial films become more standardized, evaluation of gallbladder function from such a study is going to remain confusing. I feel that if, two hours following a standard fatty meal containing cream and egg-yolk, the gallbladder shows any diminution in size and perhaps some increased density, it indicates that the gallbladder can function. So many factors govern the rapidity of emptying and the speed of absorption by the gallbladder that it is at present inadvisable to try to classify gallbladders as well-functioning, poorly functioning or nonfunctioning merely from the findings after a fatty meal.

The nonfilling gallbladder is a bone of contention. While an atrophic or contracted gallbladder or obstruction of the neck of the gallbladder or cystic duct by calculi or inspissated bile or by neoplasm, cicatrices or adhesions or external pressure may prevent the filling of the gallbladder with the dye-stained bile, it is also true that functional conditions may produce the same effect. The gallbladder which has recently been acutely inflamed or which has been irritated by a recent attack of colic, may not fill with the dye-stained bile. While spastic closure of the sphincter of Oddi from nervous or reflex causes undoubtedly exist, I believe its importance is being overemphasized. However, as I have pointed out before, a failure of relaxation of the sphincter is common and is usually due to insufficient and infrequent ingestion of foods, especially fats. In such cases the bile may become so concentrated in the gallbladder that it will not permit the dye-stained bile to enter it and no gallbladder will be visualized or, if a little does enter, the gallbladder shadow will be very faint. It is therefore important not to jump at the conclusion that nonvisualization of the gallbladder is an indication of a serious condition, especially not one requiring operation. Too often at operation such gallbladders appear to be perfectly normal. In my experience more than one-half of the cases in which the gallbladder was not seen initially, showed well filled and well functioning gallbladders after six or eight weeks of treatment such as I am going to describe. The purely functional cases, due to dietary deficiencies, showed normal or nearly normal biliary tract findings, while those due to stones or neoplasms often showed these very clearly, in a normally functioning gallbladder. Failure of gallbladder visualization after two or three attempts with a patient

known to be on an adequate diet, indicates a real pathology such as mentioned above.

Differential diagnosis has already been discussed in emphasizing the importance of a complete study. Combined cholecystographic and pyelographic studies will beautifully differentiate biliary from renal disease or disclose that both are present. Cholecystograms taken when barium is present in the stomach and duodenum or colon, will not only show their anatomical relationship but may demonstrate pressure, adhesions or fistulous openings. The frequent association of gallbladder and cardiac disease must again be emphasized.

## Treatment

The application of physiologic and hygienic principles to the treatment of gallbladder disease would seem to be most desirable and scientific and has been proved to be so by many years of practice. Yet we see in increasing numbers patients who have been treated by the unscientific fat-free dietary regime and with no efforts at prophylaxis and who have been made worse and have been subsequently operated upon unnecessarily, with poor results.

*Prophylactic treatment* should consist specifically in the promotion of normal biliary tract function and generally in the maintenance of a healthy mind in a healthy body. A normal diet, consisting of three well-balanced meals per day, preferably with two or three additional feedings between meals and at bedtime has been shown to be essential for normal physiologic health, including normal function of the gastrointestinal and biliary tracts. The caloric value of such a diet can be altered according to need, but the balance of ingredients and frequency of feedings should be maintained. Fresh air, sunshine and provision for rest and recreation are of course important. Discovery and eradication of any congenital or acquired abnormalities or diseases by periodic health examinations is too much neglected. Especially, focal infections should be prevented or cleared up if present. The role of devitalized or pyorrheic teeth, infected tonsils, chronic sinus infection, endocervicitis, prostatitis, cryptitis and other pelvic and rectal infections in the production of chronic diseases has in recent years been de-emphasized, with the result that such infections are being grossly neglected and have become an important factor in treatment of so-called "intractable" diseases. I insist upon clearing up of all focal infections not only because of their etiologic significance but to improve the general condition of the patient and to prevent complications, especially if operation is being considered.

*Physiologic treatment*: Except in very acute cases in which the type of acute abdominal calamity has not been determined, drainage of the biliary tract is most desirable. Relaxation of the sphincter of Oddi, permitting the flow of bile into the duodenum, reducing the pressure in the biliary tract, thus preventing overdistention of the gallbladder and ducts, is best accomplished by feeding of fats, preferably in the form of cream, egg-yolks and oils. Peptone, also a stimulant of biliary drainage, can be advantageously and quickly produced by easily digested proteins, such as egg albumin, the milk proteins and gelatin. So that, when a patient is vomiting excessively and is having much pain, as during an attack of biliary

colic, forced feedings at two or two and one-half hour intervals, of a mixture containing milk, dextrose, cream and gelatin, will prevent over-distention of the biliary tract and will minimize recurrent colics. I use a mixture containing one-half ounce of cream and one-half ounce of dextrose, to eight ounces of milk, with a rounded teaspoonful of powdered gelatin either dissolved in a warmed part of the mixture and then added to the whole, or mixed with water and taken before the mixture. The gelatin reduces the size of milk curds, besides supplying protein. An occasional raw egg (three per day) may be beaten into the mixture, furnishing, with the addition of some concentrated vitamins, a well-balanced, nutritious, easily digestible diet. As acute symptoms subside, bland foods may be added, until a diet resembling an ulcer diet is given. Further additions will produce a diet which I have been using in the treatment of chronic gallbladder disease for thirty years, as follows:

*Gallbladder Diet*

Breakfast: Milk, one glass
Cereal, with cream and sugar
Egg, one or two, soft boiled or poached
Bread or toast and butter
Fruit, raw, any kind

Luncheon: Milk, one glass
Egg, one soft boiled or poached, or cheese, preferably cream cheese (one-half cake)
Vegetables, cooked, all kinds
Salad, with olive oil dressing
Bread and butter
Pudding, jello, fruit or ice cream

Supper: Same as luncheon

Between meals, at bedtime and in the night if awake: one glass of milk, one teaspoonful of powdered gelatin mixed with water or the milk, and crackers, bread and butter or cake

Vitamin and mineral capsules once or twice daily.

N.B. The caloric value of this diet should be varied according to requirements. Patients may be caused to gain or lose weight.

It will be noted that no meat or meat extracts are included in this well-balanced, slightly high protein diet. The frequent association of liver damage with gallbladder disease makes it imperative to avoid these substances, which Mann and Bollman long ago demonstrated act as poisons to a damaged liver. I firmly believe that the long continued symptoms and conplications of hepatitis seen in recent years have been due largely to emphasis on the high protein diet, using meat proteins to excess. Proteins of milk, cheese, eggs, vegetables and gelatin are safer to use. After symptoms have subsided and the patient's general condition is approximately normal, meats and fish are added gradually.

The *effects* of this diet may vary. In some patients, especially in those whose gallbladders are distended due to fasting or to a fat-free diet, as for instance after an attack of biliary colic, the diet may, in the beginning, occasion considerable distress or even may precipitate an attack of colic. Persistence in the feedings in such cases will usually cause a subsidence of symptoms in a week or ten days even in patients with stones. In more than one-half of patients the diet will relieve the retrostaltic symptoms promptly, will relieve the local distress within a week or two and will greatly improve the patient's general condition. In cases which showed no gallbladder filling or poor function the good effect of the diet can be demonstrated by x-rays showing good filling and improved function. If

no improvement occurs and the gallbladder cannot be demonstrated after six or eight weeks of such a diet, complications are usually present and surgery may be required.

*Medicinal treatment*: I believe that the use of sedatives and antispasmodics is of little, if any, value and is being much overdone. Mild sedation may be necessary in the early stages of treatment in a very neurotic patient. Investigation has shown varying results from nitrites, belladonna and other antispasmodics. Excessive pains or colics may be relieved with morphine or demerol. The use of sulfonamides and antibiotics may be indicated where infection is present. Bile-acids, bile-salts and fatty acid preparations are not required and may be actually harmful in most cases. The diet should accomplish all that these preparations are supposed to do. In the presence of icterus, especially if operation is a probability, adequate doses of Vitamin K and possibly calcium should be added to the other vitamins already mentioned.

*Treatment of Complications*: In *cholelithiasis*, demonstrated by roentgen examination, operation must always be considered. In young patients under forty, in good general condition, operation is definitely indicated. After fifty and especially after sixty, if the patient's symptoms can be relieved entirely by diet, resulting in "silent gallstones", I believe in avoiding operation. If pains persist, which usually occurs as a result of pressure effects from the stone-filled gallbladder, operation should be performed regardless of age if the patient's general condition permits. During an attack of biliary colic it is better not to operate but to keep up feedings, combined with sufficient sedation to relieve the pain. With jaundice, it is usually desirable to continue the feedings, in the hope that the common duct obstruction is only temporary. If it is shown to be permanent, operation, preceded by adequate Vitamin K administration and transfusions is indicated. If no stones are found at operation experience has shown that the results will probably not be very good.

Neoplasms, demonstrated on roentgen study, are of course an indication for operation. Polyps may become malignant.

Deformities caused by adhesions or kinks, if causing persistent symptoms, will require operation.

*Complications in neighboring organs* require special attention. The diet indicated in *peptic ulcer* will also be of benefit in gallbladder disease and complications can be suitably treated. Chronic pancreatitis is also benefited by the biliary drainage as are gastritis and colitis. The appendix may at times point upward, with the tip in contact with or adherent to the gallbladder and may cause persistent symptoms requiring operation. Carcinoma of the biliary tract and pancreas may rarely occur as a result of chronic irritation, but is usually not recognized in time to be cured, although more and more radical operations are being performed for its eradication. Spinal, arthritic and neuromuscular complications require suitable care. In cases of suspected food allergy, in which sensitivity to one or more of the foods in the diet may result in aggravation of symptoms, careful diet study and elimination of offending foods is required.

*Acute cholecystitis*, as has been mentioned, is usually difficult to differentiate from other causes of an "acute abdomen". Careful collaboration between surgeon and clinician is necessary to determine whether operation should be performed. No hard and fast rule regarding indications for operation can be laid down, although it has been shown that in acute cholecystitis the mortality is lower when operation is performed after acute symptoms have subsided.

*Surgical Treatment*: In *acute cholecystitis* the type of operation depends upon the findings. It may be possible to remove the gallbladder, or, with perforation, simple drainage may be the safest procedure.

In chronic gallbladder disease, when operation has been decided upon, it should be emphasized that preoperative care is most important. Rarely is there any indication for hurry. I insist upon a careful and thorough medical regime such as I have already described, to be carried out for from two to four weeks before operation. Then the patient, with all infective foci removed, with all systemic conditions taken care of, and with the biliary tract drained by diet, can be practically assured not only of recovery from the operation but of good results afterward. Neglect of this preliminary care is the most frequent cause of poor results from operation.

At operation the surgeon determines the type of operation indicated. In general, cholecystectomy is the operation of choice. However, in my experience, when the gallbladder, though containing stones, looked so normal that the surgeon did not consider its removal advisable, results have been excellent.

*Cholangiography* during operation may locate stones or obstructions which might be overlooked by simple palpation and inspection.

*Postoperatively*, feedings should be started as early as the patient can swallow. For the first day or two it may be advisable to use only the two hour feedings of milk, cream, dextrose and gelatin mixture described earlier in this article, but as soon as possible the full gallbladder diet should be taken. The early stimulation of gallbladder drainage is most important and the large amounts of fluid thus taken by mouth obviate the discomfort of their excessive parenteral administration. Transfusion may be indicated, although with proper preparation it will usually not be necessary. The early frequent feedings usually prevent the annoying postoperative distention, encourage peristalsis and obviate the use of enemas or cathartics.

The prevention of lung collapse by early exercise, by inhalations of carbon dioxide and oxygen and by the blow-bottle, is important for the first few days.

The so-called "postcholecystectomy syndrome", usually due to inadequate pre- and postoperative care, usually responds to such treatment as described before, unless caused by overlooked or newly-developed calculi caused by stasis or by other complications.

## Summary

1. Biliary stasis is the most important causative factor in gallbladder disease.
2. Biliary drainage is largely dependent upon fat in the diet.

3. Fat is therefore important in the diet for gallbladder disease.

4. A complete general and gastrointestinal study is imperative in suspected gallbladder disease, so complications may be eliminated.

5. Gallstones are not necessarily an indication for operation.

6. Careful preoperative and postoperative care are essential to success.

### Discussion

*Dr. Horace W. Soper* (*St. Louis, Mo.*):—I thoroughly agree with Dr. Andresen that we must have biliary drainage. I also agree with him that we must avoid the introduction of bile salts in the treatment of gallbladder disease.

However, I am really astonished at the splendid results which Dr. Andresen has achieved by his method of treatment. Fifty years ago or more I first began to practice medicine, and old ladies used to tell me they formerly had gallbladder attacks but some old doctor told them to take a dose of Epsom salts every morning, and since that time they had had no attacks.

Since the publication of Timothy Læary's work on cholesterol, in 1934, I have used the diet low in cholesterol content, which means low in fat, egg yolk, which is very high in cholesterol—one of the very highest foods—and the use of internal organs, which are very high in cholesterol content. I have used that method with very splendid results. I give regular doses of magnesium sulphate, regulated according to its effect on the colon, every morning.

Meltzer, the old physiologist, whom some of you will remember, in *Gastroenterology* published his article on "The Action of Magnesium Sulphate in Producing Dilatation of the Gastrointestinal Tract." Later Lyon's article on "Biliary Drainage with the Duodenal Tube," appeared. I soon found that that method was very time-consuming, and I tried the use of magnesium sulphate by mouth, on the empty stomach. In my exhibit at the American Medical Association Convention in 1937, I demonstrated that as much bile was secured by the oral method, as by administration of the magnesium sulphate through the duodenal tube. Knowing that cholesterol is accountable and that after the age of thirty-five it slowly produces arteriosclerosis, gallstones, and cataract, which is pure cholesterol, we have kept such a patient on a low cholesterol diet, avoiding the fats, and giving the little dose of Epsom salts every morning. By that method I have secured remarkable results in cases of cirrhosis of the liver as well as in diabetes.

*Dr. Anthony Bassler* (*New York, N. Y.*):—We had a little touch on peptic ulcer this morning. I hope that is all we are going to have on it through the convention. You know, this gastroenterology subject is very interesting; it is about 75 per cent peptic ulcer and 25 per cent of everything else that usually is not supposed to be very important, and when you consider that there are some four hundred-odd clinical entities that can occur in a human abdomen and pelvis, I am just wondering whether gastroenterologists in a general way are not missing a great deal of the job which they should be engaged in and are expected to do.

The subject of gastric peptic ulcer reminds me of a story of a young boy who ran up to a policeman and asked him to come around the corner; his father

was in a fight. When the cop got around the corner, he said, "Which one is your father?"

The boy said, "I don't know. That's what they're fighting about."

That is what we have been doing for the last hundred years in peptic ulcer and nobody seems to be very much better off today. It is therefore refreshing to hear a paper like this one of Dr. Andresen's. It brings up a subject which is an old subject and a very vital one.

Now, in my clinical experience in nongallstone and many of the cholelithiasis cases, the best results that I have had have been by way of treating the intestinal canal. The suggestion has already been made to use Epsom salts, diets, purgations, and so forth.

The so-called gallbladder remedies with which we are constantly surfeited in our offices are notably unsatisfactory. To my mind this gallbladder question really starts off as a metabolic phenomenon in the liver caused by resorptive toxic products from the small intestine, and, since I have been treating my gallbladder cases from an intestinal standpoint, I feel that my results are better than by using these various proprietary remedies.

Unfortunately, most of these gallbladder cases do not remain under observation long enough to allow a very good idea of the permanency of the results; however, treated from an intestinal standpoint rather than from a gallbladder standpoint, clinically the results are not only more prdmpt and very much better, but also they are more permanent. I think the days of empirical so-called gallbladder remedies are over. I don't know how one could prove this, but I think that the empiricism that is connected with these various gallbladder remedies is so far in the air and so much beyond what intelligent people should be interested in from a clinical and from a therapeutic standpoint, that we might just as well leave them alone.

I had rather hoped that Dr. Andresen, who has done such a marvelous job in allergy, especially in connection with ulcerative colitis, might mention something about the possibility of a liver allergy. I don't see any reason why the liver could not be affected by an allergy as well as is any other tissue in the body. I hope some day he will become interested in that and see if he can produce something along that line that would be worthwhile.

The usual diets for gallbladder pathology are also notably ineffective. When cholesterol in the blood is notably elevated, an effort at fat-free dieting also is commonly ineffective. Mention has been made of high cholesterol. I think that the knockdown of cholesterol in the blood by way of diet is one of the most unsatisfactory things therapeutically that I know of in medicine. I would rather let them drink whiskey. That will knock it down quicker than diets will.

I agree with Dr. Andresen as to the use of fat in small fat meals between main ones. That is not only wise but also it has been proven by x-ray work as well as clinically. Fat-free diets in the absence of fat intolerance are based upon two fallacies, and it is rather interesting that these two fallacies have become so

popular. One is that it controls cholesterol. Well, fat does make more cholesterol to a certain extent, but certainly in the majority of instances of gallbladder pathology, elevated cholesterol is not a factor, yet, it may be, and, if it is, it deserves some attention. Secondly, it is difficult, if not impossible, to control body cholesterol by fat-free diets alone.

I have been doing quite a bit of work in cholesterol and fat-free diets and not only in cases with abdominal symptoms, but in cases of cataracts, and so forth, and with eye men, and others, and I find to knock down cholesterol by means of a diet is a tough job—unsatisfactory, to say the least. You can do something, but you don't get any nice bright results; yet, on the other hand, it is the interval, small, fat-free meals, as Dr. Andresen has brought out, which are the best cholagogue I know of, the best hepatic stimulant I know of, and the best pancreatic and duodenal stimulant I know of. They stimulate all four parts of this subject, and, taken in finely divided form, like cream, milk, butter, it is really something that we should adopt at once.

I am glad Dr. Andresen has honored us with his presence. I compliment him on the contribution. Dr. Andresen is one of our real star thinkers and clinicians in New York—even if he does come from Brooklyn—and I think that what he has brought out here is something which all of us should take home with us as the modern best way of handling gallbladder pathology.

*Dr. Rowland Ricketts* (*Merchantville, N. J.*):—Dr. Andresen is to be congratulated for his simple, down-to-earth presentation of a subject which I know is dear to most of us in this particular sphere of medicine. Also he brings us in the paper something which to me is very valuable, and that is his experience as a teacher and a clinician in over thirty years' time.

We heard very beautifully from Lord Webb-Johnson this morning, and very dynamically from Dr. Goldstein at the luncheon, that we need trained observers at the bedside, and so thirty years' experience is something which we cannot gainsay. The astute, white-coated scientist with a large sheaf of laboratory reports will not wipe out bedside observation, and I think that is one of the reasons that this paper is outstanding.

At the risk of seeming perhaps old-fashioned and naïve to you, I want to put in a very serious plug for the subject of nonsurgical biliary drainage. We used this when it was my privilege to be associated with Hahnemann Hospital, in Philadelphia, for many years, and I want to emphasize that when properly done and properly interpreted, that method gives results.

Cholecystography, of course, as has been brought out, is an indispensable procedure, but the trouble, I think, with biliary drainage is that, as busy physicians, we relegate that work to medical technicians of varying ability, honesty, and sincerity, and don't take the time to look in the microscope and see what the results are. We pick up a card and read the reports, and the reports depend upon the ability, the training, the background, and the integrity of the person who looks in the microscope, and that perhaps is one of the reasons that this method is not used as much today.

I still know of no other way in medicine to comprehend what is going on cytologically at the duodenal-biliary level than by the examination of the material which we obtain through the tube from that level.

I do not necessarily advocate that this procedure be done every day or every week, therapeutically, but I do think it should be done diagnostically at least once in the beginning, in the inception of the case, and I think it is a very excellent thing to be used periodically as a method of checking up just what is going on at that level, as we progress in the handling of the case.

I am very much in accord with what Dr. Andresen has to say about the x-ray studies. We certainly are all familiar with the old aphorism that more mistakes are made by not looking than by not knowing, but I have to put one question which applies perhaps to the private practice of medicine: Who is going to pay for all these x-rays? In organizations such as the Veterans' Administration, that is a dream. You can order what you want and get it. In research projects, x-rays are included in the budget, but I doubt whether the average man and woman on the street are able, or perhaps willing, to pay for the cost of this x-ray study. I grant you that it is necessary. I am only putting the question, and I regret that I can't give you the answer, but I think unless we find an answer to problems like this and others which have an economic background, those whose philosophy is "Something for nothing," may take the answer away from us, so it behooves us to give that a little serious thought economically as well as scientifically.

On the subject of diet, of course, we all differ. In the light of my own experience, and that of many colleagues to whom I have talked for over twenty years, I wonder whether the presentation as Dr. Andresen has given it to us is so easy, and simple, and straightforward as it would seem. I don't altogether believe it is. First of all, we have got to settle the question: Are we going to feed acute cases? Then: Are we going to feed them fat?

The draining of the gallbladder, as we all admit, will rarely ever cause rupture or even predispose to it. I think we can all agree on that, but it doesn't seem quite logical to me to require full physiological activity of an organ which is at the present minute acutely inflamed, or, let us say, quite sick. If you broke your arm, you wouldn't go out and use it. You would hang it up awhile. Should we require, by two to two-and-one-half-hour feedings, full physiological activity of an acutely inflamed organ, or should we let it quiet down awhile, as we know most of these cases will, rather rapidly, and then, when the thing is over, go on from there?

I do agree with the fat feedings, but I would much prefer in the acute attack to let the case quiet down, and when it has quieted, to build up the diet and gradually to build it back. I don't think there is any hurry about feeding in an acute case. Most of them will subside, and I don't think it matters whether we feed them tonight or tomorrow morning. The diet is not so imperative in so short a time.

Also I feel that the gallbladder's acceptance and response to fat is one of the criteria of the normal function of the gallbldder, and certainly the acute gallbladder is not normal, so we should not expect normal function and normal results from it, or try to compel it to function normally under those conditions.

One thing I am interested in—I should like to know how this diet which we have seen presented is regulated for obese people. There is a great deal of fat in it, especially if you feed people on the in-between schedule with things like milk, and bread and butter, or cake. Many of the gallbladder cases are already obese. Many of them, at least in my experience, do not tolerate fat well, and occasionally get an allergy to such things as milk and eggs. How are we going to shape up such a diet as we have seen presented, to the case which is already overweight, and yet continue the necessary feedings of fat? I should like to see some specific caloric diets on that line. I came here to learn, and that is one of the things I want to learn from this discussion.

Finally, the diet, as you noticed, mentions a very wide choice in the field of vegetables. It specifies that they be cooked. Of course, the cooking will help to soften the hemicellulose in some of the so-called gaseous vegetables, but I think it would perhaps be better to restrict that wide choice and to cut it down, and, if possible, to eliminate some of those gaseous vegetables since, as we know, flatulence is an ever-present threat to the vast majority of the gallbladder cases.

In the reference cited by Mann and Bollman I was unable to find anything as to the toxic (although I hate to use that word—"toxic") effect of meat proteins on the liver. I should like some enlightenment on that. In the composition of a diet today, of course, we recognize a need for a definite quality of protein and not necessarily quantity of protein. The meat proteins furnish the essential amino acids, as we know, and therefore have a high biological value, and in this aspect it seems to me they are superior to the vegetable protein. Probably the best food, as was recommended in the diet, is milk, and meat runs a high second.

The digestibility coefficient of meat is high. Meats yield 95 to 100 per cent of their nutritive value upon digestion. Their proteins are of a high biologic order. Legumes and potatoes contribute only about 80 per cent of their proteins, so why not use meats? I really would like to put that question.

As to gelatin, if the gelatin is given merely to be digested to peptone stage for biliary stimulation, I am all for it. Gelatin as a protein is a deficient protein and therefore will not put or keep the patient in nitrogen balance. It will assist other proteins, but of itself it is deficient.

As to the adequate postoperative care, that is something I think that we should all stop to think about.

An Italian woman who came to me told a story like this: She had had her gallbladder removed on a most nebulous group of symptoms. I finally found out, in talking to her later, that there was difficulty with her husband at home, and the woman said to me, "if you told me to have my leg off, I would go in the hospital and have it off, just to get away from my husband." Someone had talked

her into having her gallbladder out, and she had had it done. On the sixth postoperative day the surgeon patted her on the back and said, "You are cured. Now you are well. Go home and enjoy a good spaghetti dinner," which she did, and when she saw me next morning, she was still vomiting copiously.

Now, I don't know how we are going to do it, but somehow, and at some time in the future, we have got to get into the minds of patients, and of a certain group of doctors, that the patient is not cured of the disease when the operative wound is healed. I always tell my patients in any surgical case, "Now that you have recovered from the operation, you are in a position to get well, but the operation in itself is not your total cure." I think if we can send them out in that way, we will get better cooperation from them afterward, with far better results as the case progresses.

Finally, I think it is in keeping with the high quality of Dr. Andresen's work, although perhaps he did not mention it in his address—but it is written in the paper—that he has one short, simple sentence on the use of sulfonamides and antibiotics. It is a good thing, I think, to see a doctor who is oriented in his field and who is not merely shooting in the dark.

*Dr. Albert F. R. Andresen* (*Brooklyn, N. Y.*):—I am grateful and pleased that my presentation provoked such interesting discussions, and that at least one discusser agreed with my statements as to the importance of fat in the diet. About 35 years ago when I was dissatisfied with the use of various salts, with their cathartic, dehydrating, exhausting effects, in the treatment of biliary tract disease, a study of the physiology of this tract convinced me that frequent feeding of fats would promote biliary drainage and be of distinct advantage in any disease of this tract. I tried it and it worked and for 35 years I have used and advocated this treatment and have found it unnecessary to use any medication except in acute cases to relieve pain. When the gallbladder is acutely inflamed it is not to be expected that it could contract enough to do it any harm or good, but the feeding of fats, by promoting physiologic drainage of the biliary tract, tends to lessen the pressure in the tract and thus tends to reduce the probability of rupture. I have used it regularly and have had only good results. The same is true of early frequent feedings postoperatively.

The use of fats and the frequent feeding schedule does not necessarily put on weight. It is a simple dietetic problem to plan a diet of the proper caloric value. I always plan to have the patient attain and maintain a normal weight. This often necessitates a considerable reduction. I remember one patient I reduced from 350 to 250 pounds during such feedings.

I am surprised at the general neglect of the principles established by Mann and Bollman and their associates in the 1920's, when they demonstrated the harmful effects of animal proteins and their extractives on damaged or partially extirpated livers. I believe it is this neglect which has resulted in the high morbidity in hepatitis in recent years, when the need for a high protein diet has been met by excessive feeding of meats. I know that avoidance of meats is beneficial. High protein values can be maintained in a diet of eggs, high protein vegetables,

gelatin, and particularly cheese. The addition of choline and methionine is unnecessary when much cottage cheese is fed.

In regard to hypercholesterolemia, there has been no proof that the amount of fat in the diet has any regulatory effect on blood cholesterol, and it is also true that there is no proof that the quantity of fat ingested influences arteriosclerosis or the formation of gallstones or cataracts.

In regard to diagnosis of biliary tract disease, in its day Lyon biliary drainage provided a valuable method, but today, when not only the gallbladder but all of the ducts can be visualized by cholecystography, such drainage is usually unnecessary. As to the cost of x-rays, that is a serious matter, but when a patient is faced with the possibility of operation, the additional expense involved in finding out definitely all that is the matter with the gastrointestinal and biliary tracts is negligible and may prevent ill-advised operation or disclose conditions which urgently require operation. I always insist on a complete gastrointestinal study even though gallstones have been shown to be present.

# EXPERIENTIA DOCET*

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I thought I would much rather talk to you than read an address. If you haven't got a prepared text, although you talk more freely you get more into heart to heart touch with those of your audience who remain awake.

I have just come from Chicago, where I have been talking to the American College of Surgeons on "Science in Surgery", and today I would like to say something on that subject, emphasizing that Surgery is an art as well as a science, and that it is primarily an art in many respects.

We flatter ourselves that we live in an age of science, and that we are supreme in this respect in the history of the world, but if we look back a few thousand years we find that scientific methods, and the scientific approach, were the intellectual habits of the Greeks. Then the Dark Ages came, and it looks at the present day as if there is a danger of the world going through yet another period of darkness.

There is a great force active in the world, poisoning men's minds, that may put culture and science back, as they were put back once before, for a long period. It is a matter for very serious thought that when these revolutionary ideas infiltrate great communities, the first action of the leaders when they achieve a coup d'état, is to attack those who have been trained in scientific methods, those who are accustomed to independent thought and are not easy victims of mass propaganda.

You have seen examples in many countries in Europe, where the first attack is made on the professional classes, the lawyers, the scientists, the doctors and the Church.

That is rather a political digression, but it has to do with the Dark Ages, and the Dark Ages, when they came before, lasted ten centuries, and in the revolution of those ten centuries, in the words of Gibbon: "not a single discovery was made to exalt the dignity or to promote the happiness of mankind".

But, let us turn to the brighter period that followed these ten centuries of darkness, to the Renaissance, when the great anatomists laid the foundations for surgery and for medicine.

The work of the great anatomists sounded the reveille, and the dawn was hailed by Harvey. He compelled a return, after ten centuries, to the scientific methods of the Greeks, the experimental method. And the experimental method is, beyond all shadow of doubt, the most effective implement for the advancement of knowledge ever invented by man.

What were the gifts that Harvey required to revolutionize thought and bring the world back to the scientific method? In the first place, constructive imagina-

*Read before the Fourteenth Annual Convention of the National Gastroenterological Association, Boston, Mass., 24, 25, 26 October 1949.

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tion; in the second place, the power of deduction; in the third place, most rigid self-criticism; and ultimately such patience in labor as has seldom been equalled.

His objective was to search and study out the secrets of nature by way of experiment, and there is great inspiration in his advice to the young man: *"Dei laboribus omnia vendunt"*—"For toil the gods sell everything". Harvey tore down that which was obvious rubbish, but he laid no profane hands on symbols in which the spirit of truth lingered. As I said the other night in Chicago, in this respect he showed more sense than his great contemporary, Cromwell, and, he showed more sense than some modern politicians who, when evolving new plans are inclined to tear things down instead of building on the old foundations and holding fast to that which is good.

Harvey was followed by Hunter, who founded scientific surgery. That is the inscription on his tomb in Westminster Abbey, "The Founder of Scientific Surgery," and that epitomizes the man's life and his contribution to the service of humanity.

There are other interesting inscriptions on stones in Westminster Abbey. One of the most attractive, I think, and one which reflects most fully affection, admiration, and esteem, is the simple inscription on the slab which marks the place of Ben Jonson—four words: "O rare Ben Jonson!"

I will give you, Mr. President, a copy of the letter, an exact copy, an exact replica, of the letter which Hunter addressed to Edward Jenner, in answer to an inquiry, "What shall I do? I think so-and-so." In Hunter's reply is the famous phrase, "Why think? Why not try the experiment?"

I am naturally a devotee of Hunter, because his great museum was entrusted to the Royal College of Surgeons, over which I had the honor to preside for eight years. But his immortal fame does not rest on his great museum. The reason for his enduring reputation is that he was a consistent and expert user of the experimental method. That is why he is a quickening influence among surgeons today, and that is why Sir James Paget, in a Hunterian Oration, used the following eulogy:

"The influence of such men as Hunter reaches far beyond the time and space of their conscious activity. Their true thoughts live after them. Thus, when we honor the memory of Hunter, we honor not only that which is past but that which is still present, a still abiding power, doing good."

Hunter made a scientific approach, founded scientific surgery. Lister, his greatest follower, achieved his great results because he had a scientific training. He had a scientific approach to the problems of the time and was able to see the truth and the possibilities, the implications of Pasteur's discoveries.

But now, having paid tribute to science, indeed having paid homage to science, it is well to remember that medicine is more than a science. It is also a practical art.

Surgery is in large part a practical art. The surgeon is not in the same position as the scientist, who must have proof before he acts. In many cases the surgeon has to act at once. He cannot wait for factual proof. He is the heir to all

the advances in the practical art which have accumulated through the centuries. He has to use the aptitude which he has cultivated. The particular case is his problem and the patient is relying on him. His intuition will lead the master clinician to diagnosis perhaps before all the facts are before him, but intuition is only experience called back into the mind, though not deliberately. The surgeon can often stand confidently between life and death. He can often hold death back even as it is about to strike, if he has knowledge, judgment and skill.

And yet, the master of this practical art lives only in his time. He is like an actor, a prima donna, a prima ballerina, or a matador—in the words of Shakespeare, he

". . . . . struts and frets his hour upon the stage

And then is heard no more."

But his work is not lost. Every logical advance that he has made will be carried on.

But, mastership of the art alone, equipment with the factual knowledge alone, is not enough. The doctor must have had his intellectual curiosity aroused by his scientific training and, if he has that, then he will understand; he will be on the lookout; he will make observations, whether he is trained to work in the laboratory or whether his life is to lie in the clinical field, where he may still make observations of fundamental importance.

All advances do not result from laboratory experiment. Any doctor in any community, however small, and however small his opportunities, may make a fundamental discovery. You have only to recall Edward Jenner, to whom that letter was addressed by John Hunter. This general practitioner down in a little village in Gloucestershire, in England, watching, looking, observing, thinking, and concluding that if the milkmaid who got cowpox did not get smallpox, there must be some connection between the two.

In the manuscript diaries of Joseph Lister, which we preserve reverently in the Royal College of Surgeons of England, there are records that when the mold of penicillium grew on his culture media, Lister noticed that the growth of bacteria was interfered with. But he also recorded in the diaries significant speculations as to why the bacteria did not grow in the neighborhood of this mold. That was Lister, 'way back nigh on eighty years ago, and his records are there. The man was thinking.

The bacteriologist Sir Alexander Fleming, seventy years later made the same observation and reflected:—"This may be valuable in treatment." That was observation. It wasn't experiment.

Then the experiments began, and then the chemical constitution of the mold penicillium was analyzed and then, after many years of patient work, penicillin was isolated from the mold by Sir Howard Florey.

Another possibility for observation outside the laboratory in practicing the art of medicine is by keeping careful records and by the study of those records to arrive at conclusions which may be of great significance and value. There was

the method of Louis, in Paris—the study of large series of cases. Louis was the greatest French medical teacher when the reputation of the Paris School was at its height. That method has been followed, I think perhaps too assiduously, in this country, and a clinician often feels that unless he has enormous numbers of cases, he will not be able to draw valuable conclusions; but that isn't true.

Wilfred Trotter, with a few cases, was able to draw conclusions in regard to pharyngeal growths, which have stood the test of time—careful observation in a few cases.

Now I was born the year that Bend Or won the Derby, so now you know how old I am. If that doesn't convey anything to you, I was born in the year that a surgeon Sir Henry Morris of my hospital, the Middlesex Hospital, first had the temerity to open the intact kidney and extract a stone. Anyway, it is not so long ago. As a young graduate I worked with Sir William Thorburn, in Manchester, and became his Chief Assistant. Later I crashed into London and refused to be crowded out, but when I was assistant to William Thorburn, I went with him to a small manufacturing town in Lancashire, to operate on a case of appendicitis, and there I met James Mackenzie, the patient's family doctor. James Mackenzie, the founder of modern cardiology, was working as a general practitioner in Burnley. Modern cardiology is based on his observations of a few cases in a comparatively small community examined by the polygraph made by the watchmaker at the corner of the street. That is enough to stimulate any individual, no matter where he may be placed. He must not think that he has only to carry on with his routine work. He has got to realize that he has the opportunity of making observations which may be of help to his fellow-practitioners and to humanity.

There is no limit to it, and I would like to give you an example which has often been quoted of how science may lag behind clinical observation. Linde, in 1754, observed that men in sailing ships, suffering from scurvy, were relieved of their symptoms if they were given lemon juice and lime juice. It took forty years for the British Lords of the Admiralty to make it a rule that their sailing men-o'-war (marauding your coasts and interfering with your commerce), should carry lemons and lemon juice, and thus avoid the loss of personnel, which was far greater from scurvy than it was from guns of opposing fleets.

Linde's discovery was a practical fact, the result of observation, a fact that was stored and used. There was no scientific explanation. That did not come until one hundred and fifty years after Linde's observation when the physiologists discovered Vitamin C.

So, wherever your path may lie, reckon that you have opportunities for advancing the science and practice of medicine.

I should like just to quote to you the remarks of a great British surgeon, Wilfred Trotter, a Fellow of the Royal Society, the highest hallmark that a man can achieve. It is the proudest award he can get, F.R.S., Fellow of the Royal Society. Only two surgeons have ever been President of the Royal Society—Sir Benjamin Collins Brodie, and Lord Lister. That gives you a measure of the calibre of this select company.

Wilfred Trotter wrote as follows:

"Now that the prestige of science is so high, the statement that a great part of medicine still retains the status of a practical art, is often made with a note of apology."

"Nothing could be less justified by a realistic sense of cultural values. The method of the practical art was the first instrument forged by man for the subjugation of chaos. At the dawn of civilization the preservation of knowledge was far more important than its discovery. The accidental fruits of experience and the creations of genius could be saved from an infallible oblivion only by being preserved in the precepts and traditions of a practical art. It seems probable that in the practical arts man made his first serious attempt at the continuous storage of knowledge and the establishment of a progressive culture."

When your president insisted on me giving a title for this address I was overwhelmed with work. I eventually consented, however, and selected "Experientia Docet". I thought I could jump off from that into any theme that my fancy seized upon at the moment, and my theme today is that there is no opposition between the science of medicine and the curative art.

The more science we have, however, the greater seems the need for human values, and each advance in technic makes personal healing more essential. A good doctor must have many skills, and one of the skills which is likely to be neglected in the developments of the present day is the skill which comes of devotion to his responsibility to his patient.

I listened to an address recently. I won't say who delivered it, but the lecturer shocked me when he said that the duty of the surgeon was to treat the patient's disease. He has got to do more than that. He has got to treat the patient. If he is not a good doctor, if he doesn't carry his patient and help him to bear his burdens, what is the logical result? The psychologist arrives on the scene and says, "You don't understand how to treat patients. You may know the disease; you don't know about patients." But a good doctor carries his patient, and helps him over the stiles.

What I want to make clear is that to handle disease, or to handle the patient on the physical plane alone is to deal with only the half. The cure of the sick sometimes depends on kindness more than efficiency. The patient is sick—he wants his symptoms relieved. He wants comfort. He hopes for cure. He may think you have given him cure, but at least you have given him something of yourself if you are a good doctor.

First, the good doctor must have the power of attention, to give, and to make the patient feel that he is giving, his whole mind to the problems which mean so much to the patient. Secondly, he must have intuition, the power to draw inferences from experience. Thirdly, he must develop the art of handling living flesh and living tissue with gentleness and sympathy, and without causing pain, or, in the unconscious patient, without causing unnecessary damage or shock. Lastly, as I have said, he must learn the art of handling the sick man's mind, or he is not a good doctor.

Ladies and gentlemen, I hope I have interested you. I trust I have not wearied you. I have loved meeting you. I hope to see more of you, and I thank you for your kind welcome and patient attention.

Discussion

*Dr. Arthur W. Allen (Boston, Mass.)*:—It would be presumptuous of me to try to discuss this learned discourse which you have heard this morning. I have known Lord Webb-Johnson for a great many years. I know of no one individual who, to my mind, could better have exemplified the best in medicine and surgery in this world, this morning. He has the faculty of putting before us in such an attractive manner, important facts regarding our profession and its duties to humanity, that it would be impossible, I think, to adequately give him his just due.

I was extremely interested that he chose to stress the art of medicine and surgery this morning. He had not confided in me just what tangent he might take. I have an idea that perhaps the notes from which he spoke were written early this morning when he probably should have been sleeping.

I have been alarmed at the present trend in medicine and in surgery regarding the stress on the laboratory, on the scientific side, to the exclusion of what Lord Webb-Johnson chooses to call the art of medicine and surgery. You have all seen it. Doubtless many of you have been as worried about it as I have been, but, to look upon the patient as you might, if you were callous, look upon one of the dogs in your laboratory—and the affection that we have for these animals, I am sure, should be stressed—but, actually they are numbered—sometimes they are named—and too frequently I have seen patients who were treated much the same way. This is wrong, gentlemen, and when you get an opportunity to read these remarks, by Lord Webb-Johnson, you will read so much between these lines, that I am sure you will see adequately the point he tried to make.

Every doctor has got to be a humanist. Every doctor has got to be somewhat of a psychologist. Once I overhead a surgeon say, after he had watched a very beautiful surgical procedure, a complicated bit of technic, "This operation was performed exactly like an artist paints a picture." Now that is not what Lord Webb-Johnson means by "the art of surgery". There are plenty of good technicians in this world. What we need is more stress on the human relationship, and that is something that we are close to losing in this country, not only through our perhaps overemphasis on the scientific side, but also because we are close to having interference that will affect us all.

I have enjoyed tremendously listening to Lord Webb-Johnson this morning, and I think probably you all have enjoyed it as much as I have, but I have never heard him speak—and I have heard him many times—when I thought that he conveyed a greater message than he has this morning.

*Dr. William W. Lermann (Pittsburgh, Pa.)*:—First as an individual, secondly as a citizen of the country, thirdly as a practicing physician of the country, fourthly as a member of this organization and an officer, I wish to express to you, sir, my sincere thanks and appreciation for your presence here today and the interesting and instructive messages that you have conveyed.

It would seem to me, very frequently the unspoken word may mean as much as the spoken word. I know that the doctor has had a world of experience which has been professional as well as otherwise, and I again wish to thank you for being with us and alerting us to some of the problems of today.

# VAGOTOMY: AN EXPERIMENTAL AND CLINICAL APPRAISAL OF THE EFFECT OF SECTION OF THE VAGUS NERVES AT DIFFERENT LEVELS

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and
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It has long been known that section of the vagus nerves at different levels produces different effects on the esophagus. The effect of total vagotomy can only be attained by severance of the vagus nerves in the neck. In 1932, 1933, 1940 and 1948 we carried out numerous experiments in animals on the effects of section of the vagus nerves. During this work we have repeatedly noted that cutting the vagus nerves at different levels produced varying effects upon the esophagus. The purpose of this communication is first, to direct attention to the varying differences encountered in cutting the vagus nerves at different levels; secondly, to emphasize that esophageal symptoms which occur following vagotomy of the lower end of the esophagus may be due to a traumatic esophagitis; and thirdly, a comparative study is made between the experimental and the clinical experiences encountered in 30 cases of vagotomy done in the human.

Nearly all of the older information pertains to the physiologic effects of electrical stimulation and vagotomy on the cervical vagi. In experimental total bilateral cervical vagotomy, the esophagus immediately begins to dilate, and in a brief period of time, the lumen becomes two or three times its normal caliber. Besides producing a true achalasia of the end of the esophagus, the stomach also becomes dilated and atonic, similar to that which is found after abdominal vagotomy. Our experimental work is in accord with others, who have shown experimentally the differences in effect of severance of the vagi at different levels.

In abdominal vagotomy, true signs of cardiospasm are rarely observed. There is no dilatation of the lumen of the entire esophagus with atony as compared with the findings obtained in total vagal severance in the neck. Severance of the vagus nerves in the abdomen does not invariably produce a spasm at the cardia end of the esophagus. Soon after abdominal vagotomy there is, at times, a transient spasm or irregularity which tends to disappear. When the vagi are severed at a lower level transthoracically or intraabdominally its physiologic effect will be noted only in the stomach. The esophagus is not ordinarily affected. Owing to the mutilation, trauma, and extensive manipulation of the lower esophagus, which is occasionally necessitated in the operative procedure of severing the vagi and its branches, some esophageal abnormality will be noted at times, but this has only been temporary phenomena. There can be no question that the extensive traumatization of the lower esophagus during vagotomy is responsible for the production of an esophagitis, causing esophageal symptoms.

This experimental work was done at the University of Maryland, School of Medicine under a grant provided by the Doctor Julius Friedenwald Experimental Fund.

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At Sinai Hospital, Gann and others have performed vagotomy in 30 cases; 10 were intrathoracic and 20 intraabdominal. A study of these cases showed an absence of symptoms referable to the esophagus. Postoperatively, they have not shown any roentgen signs of esophageal abnormality, except for occasional early transient spasm at the cardia end.

The mechanism of cardiospasm or achalasia is not clearly understood. It is known, however, that in this condition there is an imbalance resulting in a failure of the cardia end of the esophagus to relax. Autopsy specimens have shown a degeneration of the intrinsic nerve plexus in the lower esophagus. Many years of

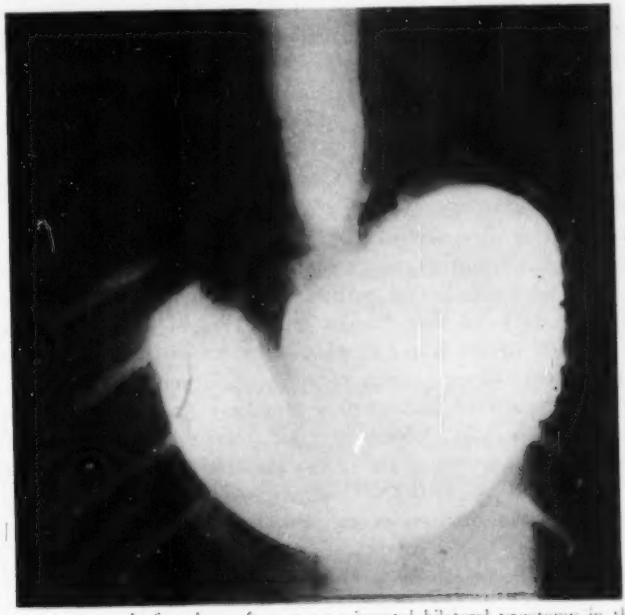

Fig. 1—Roentgenogram made five days after an experimental bilateral vagotomy in the neck. Illustrates a dilated esophagus with a smooth tapering constriction at the cardia end, characteristic of an achalasia. The stomach is shown to be markedly dilated and atonic.

experimental investigation on the vagus nerve have proven that this nerve plays a very significant role in the etiology of cardiospasm.

Bernard, Schiff, Chauveau, Kronecker and Meltzer established experimentally that vagus section of both nerves produces a closure of the cardia end. Sinnhuber found that cutting the vagi in the neck has a different effect than when the vagus nerves are cut above the diaphragm. Kure cut both nerves in the neck, found that the animals died of hunger because of their inability to swallow, but when the nerves were cut just above the diaphragm he could not produce any spasm of the cardia, the animals remained alive without developing any esophageal dilatation.

Kronecker and Openchowski were able to produce both relaxation and contraction of the cardia by induction current stimulation of the vagus. They believed

one part of the vagus produces relaxation, another part contraction. Langley also investigated the selective action of the vagus nerve. Rieder found that electrical stimulation of the upper vagus always produces contraction of the esophagus and cardia. He could not produce these effects by sympathetic stimulation.

It is well known that experimental severance of both vagi in the neck will result in death as a result of its effect upon the cardiorespiratory organs. When cut below the cardiac branches, transthoracically or intraabdominally, there is no effect upon these organs. In our experimental study both vagus nerves were sectioned in the neck of five dogs. Large portions of both nerves were removed. Though all of the animals ultimately died, they remained alive from three to fifteen days when placed on a milk diet. Roentgenologic examination of the esophagus revealed a definite contracture at the cardia end, the degree of contracture and dilatation of the esophagus increasing with the length of time the animal survived. All of the animals showed the characteristic signs of cardiospasm, with dilatation of the lumen, though none showed evidence of complete obstruction. Besides the esophageal changes, the stomachs were dilated and atonic revealing marked gastric retention.

The fact that cardiospasm can be produced by severance of the vagi in the neck and not by low transthoracic or subdiaphragmatic vagotomy, would indicate that there must be a number of pathways through which the vagus nerve acts. It is clearly demonstrated that the cut cervical vagus nerve produces an unbalanced nerve action on the lower esophagus by way of the sympathetic nervous ganglionated system and/or by way of an intrinsic nerve mechanism. Since postmortem studies of patients with cardiospasm have demonstrated degeneration of the intrinsic nerve plexuses of Meissner and Auerbach and since resection of the vagus nerves result in spasm of the cardia end and atonicity, the conclusion may be drawn that disturbance of the sympathetic nervous system plays an important primary role in the mechanism of cardiospasm. Severance of the vagus nerves causes the impulses to travel only through the ganglionated cord and this imbalance is associated with an achalasia. Our experimental work suggests a mechanism by which the severed vagi in the neck produce cardiospasm.

## Conclusions

1. The effect of vagus section depends in a large measure on the level at which it is severed. Total effect of vagotomy can only be secured by severing the vagus nerves in the neck. This is usually a fatal procedure.

2. Section of the vagi in the neck experimentally produces a contraction of the cardia end of the esophagus with all of the characteristics of cardiospasm or achalasia.

3. On the basis of our experiments it appears that section of the vagi in the neck produces an imbalanced autonomic nervous mechanism with preponderance of action through the the ganglionated cord, resulting in a failure of the cardioesophageal opening to relax.

4. Transthoracic and intraabdominal section of the lower vagi do not materially affect the lower esophagus. This is borne out experimentally and in human

subjects. A temporary contraction or disturbance of the lower esophagus may be observed following vagotomy in human subjects, but in our experience true cardiospasm is not produced by intrathoracic or intraabdominal vagotomy.

5. The esophageal symptoms occasionally encountered following intrathoracic and intraabdominal vagotomy is probably due to a traumatic esophagitis.

6. In our clinical studies of 10 intrathoracic and 20 intraabdominal vagotomies, there were no demonstrable instances of cardiospasm.

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# SURGICAL REPAIR OF FEMORAL HERNIA*

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## Historical Background

Three methods of repair of the femoral hernia are commonly used:

1. *Bassini's method*, published in 1890. The incision is made below Poupart's ligament, the sac is tied off as high as possible, two or three interrupted sutures are passed backwards through Poupart's ligament and the pectineus fascia at

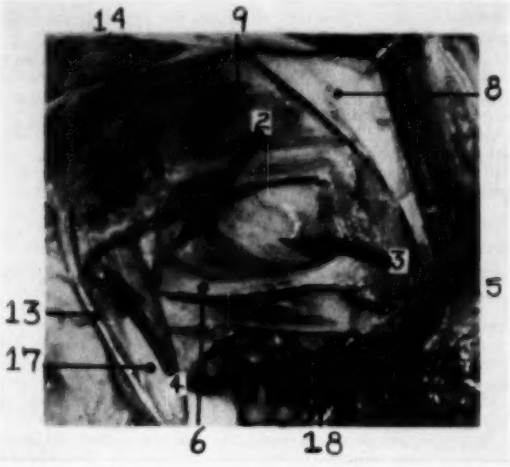


Fig. 1—(Dissection)—External oblique aponeurosis incised, exposing the transversalis fascia; **2**-transversalis fascia; **3**-conjoined tendon; **4**-abdominal ring; **5**-region of the pubic spine; **6**-Poupart's ligament; **8**-upper margin of incised external oblique aponeurosis, reflected medial; **9**-internal oblique muscle; **13**-ilioinguinal nerve, lying on the spermatic cord; **14**-iliohypogastric nerve, crossing the internal oblique muscle to enter the rectus sheath; **17**-spermatic cord, lifted from its bed; **18**-undescended testicle.

the pectineal line and include Cooper's ligament if possible. After tying these, a second row of sutures approximates the falciform process and the pectineus fascia.

2. *Halsted's method*, published in 1890. The approach is above and parallel with Poupart's ligament, the cord is lifted from its bed, the transversalis fascia is incised. The femoral hernia sac is freed and delivered into the wound, opened, tied off and transfixed. The transversalis fascia is closed and the medial aponeu-

*Presented at the Postgraduate Course in Gastrointestinal Surgery of the National Gastroenterological Association, Boston, Mass., 27, 28, 29 October 1949.
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rotic layers sutured to Poupart's ligament and the cord transplanted subcutaneously.

3. *George Lotheissen's method*, published in 1898. Cooper's ligament was used as the inferior and posterior anchor for the medial aponeurotic layers in 12 cases.

Anson, Professor of Anatomy at Northwestern University and Chester B. McVay, from 1939 to 1942, published the results of their careful dissection of 125 cases, showing that many congenital anomalies and deficiencies existed in the fascial layers of the inguinofemoral region of the abdominal wall, capable of producing multilocular herniae. They repopularized the technic, first advocated by Lotheissen, using Cooper's ligament as the inferior and posterior anchor for the transversus abdominus aponeurosis and transversalis fascia from the pubic spine to the femoral vein. This repair of the posterior inguinal wall was advocated in all types of herniae in the inguinofemoral region, except those small indirect herniae of less than 2 cm. Harkins and Swenson, at the Henry Ford Hospital in Detroit, supported this view in some 300 cases of inguinofemoral herniae with only a few recurrences.

TABLE I

174 Femoral Hernia
Boston City Hospital
1940—1948

| | | |
|---|---|---|
| Male | 72 | 42% |
| Female | 102 | 58% |
| Right | 110 | 63% |
| Left | 58 | 33⅓% |
| Bilateral | 6 | 3⅓% |
| Average Age | 60 years | |
| Living Cases | 57½ years | 146 |
| Death Cases | 73½ years | 28 |

In view of the recent discussions of femoral hernia repair, appearing in the literature, we have reviewed the controversial anatomy and our experience with femoral herniae at the Boston City Hospital. It may also be of interest to consider why hernia, particularly femoral, should concern the gastroenterologist. This becomes apparent when it is shown that we are dealing with strangulation, incarceration, obstruction, peritonitis, and resection.

## McVay's Cooper Ligament Repair

*Skin incision*:—This is made above and parallel with Poupart's ligament from 2 cm. below the anterior superior spine of the ilium to the pubic spine (Halsted) or McVay's incision in the skin lines for cosmetic effect from the internal abdominal ring horizontal to within 1 cm. of the midline.

*External oblique aponeurosis*:—This is incised with the knife 1 cm. above the external inguinal ring, then extended superiorly to avoid the ilioinguinal nerve lying on the cord, and the iliohypogastric nerve crossing the internal oblique muscle to enter the rectus sheath. The medial and superior flap of aponeurosis is dissected medially to the rectus sheath. Poupart's ligament is exposed and the spermatic cord, or round ligament, is delivered from its bed and retracted with a tape (Figure 1).

*Transversalis fascia*:—which lines the peritoneum of the abdominal wall, is exposed. The conjoined tendon, formed by the fusion of the internal oblique aponeurosis and the transversus abdominus aponeurosis, lies anterior to the transversalis fascia and medial to the cord (Figure 1). McVay states, "femoral hernia is a defect in the posterior inguinal wall by virtue of the insertion of the posterior inguinal wall into Cooper's ligament".

Cooper's ligament, described by Sir Astley Cooper in 1804, is exposed by incising the transversalis fascia and cleaning away the areolar and fat tissue over the superior ramus of the pubis, from the pubic spine to the femoral vein (Figure 2).

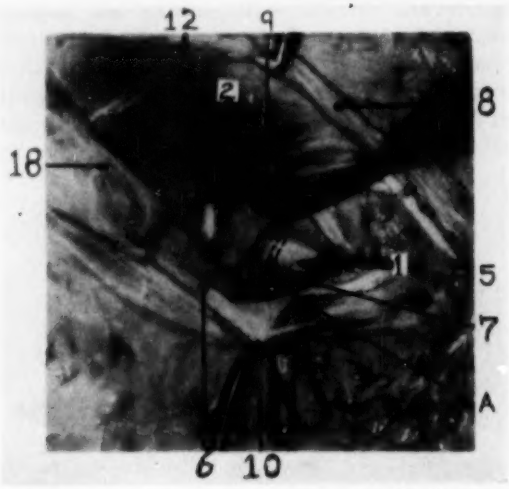


Fig. 2—(Dissection)—Transversalis fascia incised to expose Cooper's ligament. Silk loop represents the direction of a femoral hernia. Cooper's ligament extends from the pubic spine posteriolateral along the iliopectineal line. **1**-Cooper's ligament; **2**-upper leaf of incised transversalis fascia, retracted superiorly to expose more of Cooper's ligament; **5**-region of pubic spine; **6**-Poupart's ligament, retracted inferiorly; **7**-Gimbernat's ligament, inner 1.75 cm. of Poupart's ligament reflected posteriorly and attached to the subadjacent iliopectineal line; **8**-upper margin of incised aponeurosis of external oblique, reflected medial; **9**-internal oblique, muscle; **10**-femoral vein; **12**-silk thread around the inferior epigastric vessels; **18**-undescended testicle with cord, reflected superiorly; **A**-location of the variation of the obturator artery at the lacunar edge of Gimbernat's ligament, when it is a branch of the inferior epigastric artery in about 15 to 35 per cent of the cases.

*Boundaries of the femoral ring*:—These are Poupart's ligament and transversalis fascia, anteriorly; Cooper's ligament on the superior ramus and the pectineus muscle and fascia, posteriorly; femoral vein, laterally; lacunar edge of Gimbernat's ligament, medially. In about 28 per cent, the obturator artery may arise from the inferior epigastric artery, and pass along the lacunar edge of Gimbernat's ligament, thus almost encircling the neck of the femoral sac. It should be ligated when present, and Claude S. Burton also advises tying off the inferior epigastric vessels to effect a better repair.

*Incarceration and strangulation of femoral hernia*:—If a femoral hernia extends only through the femoral canal it is termed incomplete, and is small, due to the resistance of the surrounding tissues and is difficult to detect, especially in the obese person. The complete or large hernia projects into and through the fossa ovalis (Figure 2). Stricture of a hernia may occur at the neck of a sac, or in the fossa ovalis, but usually occurs where the falciform process joins the free edge of Gimbernat's ligament. In large charitable hospitals, such as the Boston City, 66 per cent of the femoral hernias may show incarceration or strangulation on admission (Table II). This is due mainly to the reluctance that this type of patient shows in appearing at the hospital until the symptoms are severe enough to look for relief from pain.

*Femoral sac and contents*:—The femoral sac and contents is delivered into the wound, thus converting it into a direct hernia. To release from the stricture which may prevent the delivery of the sac, it is often necessary to invert the point of a knife and incise Gimbernat's ligament, anteriorly and medially. The sac is opened, the contents inspected, and gangrenous bowel, if present, is resected, then the sac is tied off at the level of the peritoneum.

TABLE II

OPERATION—162

SURGEONS—100 (ESTIMATED)

| | | |
|---|---|---|
| Above Poupart's Ligament | 84 | 52% |
| Below Poupart's Ligament | 78 | 48% |
| Reducible Not Op. | 8 | |
| Inoperable (died) | 4 | |
| Recurrent Femoral | 2 | |
| Previous Oper. Inguinal | 8 | |
| Strangulated | 51 | 31½% |
| Incarcerated | 55 | 34½% |

Average Days in the Hospital—16½

*Cooper's ligament repair*:—Passing a finger laterally along Cooper's ligament and the superior ramus of the pubis about 4 cm., lies the femoral vein, which can be protected either by the finger or with a short one inch Dever type of retractor. Sutures are passed from the superior leaf of the incised transversalis fascia, and the conjoined tendon through to Cooper's ligament, from the femoral vein to the pubic spine. When these sutures are tied, the femoral ring is excluded from abdominal pressure by newly formed anterior abdominal wall (Figure 3). Sutures are passed from the transversalis fascia, anterior to the femoral vein, through the reflected edge of Poupart's ligament to close off the space anterior to the femoral vein (Figure 3). To relax the tension of the superior leaf of the repair, an incision may be made in the anterior rectus sheath, beneath the aponeurosis of the external oblique 1 cm. above the pubic spine superiorly, for approximately 3 cm. (Figure 3). In many instances, this procedure may not be necessary. This is particularly true in the female because of the ease with which the upper leaf approximates Cooper's ligament.

McVay returns the cord or round ligament to its bed, sutures the external oblique aponeurosis, and then the skin to complete the repair. Some authors, such as Claude S. Burton, feel that the aponeurosis of the external oblique should

be included in the first row of sutures and use two strips of fascia lata as continuous sutures. As the femoral vein is approached, the line of suture is brought above the femoral vein and is re-directed toward the spine of the pubis. It includes the edge of Poupart's ligament, Cooper's ligament, and imbricates the external oblique aponeurosis to fill in the triangular space, Burton's trihedral space. Burton transplants the cord subcutaneously and closes the skin. Thus Burton's repair is a combination of a McVay, Gallie and Halsted (Figures 3 and 5). The principle of the McVay repair is extremely logical in that the intraabdominal pressure is shunted over the brim of the pelvis into the pelvic cavity, instead of directing excess stress into the inguinal recesses. The repair is particularly effective if the reconstructed anterior wall is of adequate strength. In many

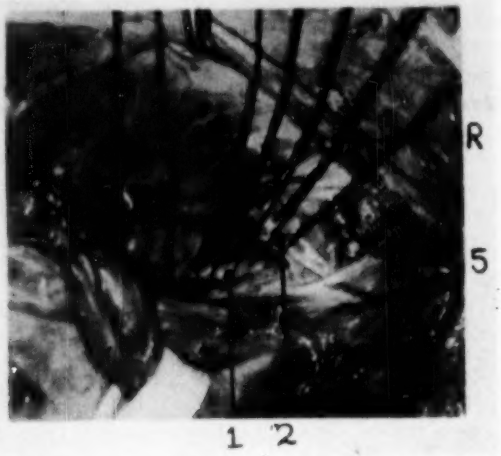


Fig. 3—(Dissection)—Silk sutures placed between the conjoined tendon and the transversalis fascia and Cooper's ligament. The two lateral sutures are placed between the transversalis fascia and the inferior edge of Poupart's ligament. External oblique aponeurosis lifted to make relaxing incision in rectus sheath just lateral to the fusion of the external oblique aponeurosis with the rectus sheath. **1**-Cooper's ligament; **2**-upper leaf of transversalis fascia; **5**-region of pubic spine; **R**-relaxing incision in rectus sheath.

instances it has been found to be totally inadequate, due to the poor quality of the transversalis fascia as well as conjoined tendon, in which case it offers a very poor restraining wall. On the other hand, it is generally found that females possess a very good fascia and it is in this sex, that the frequency of femoral herniation statistically favors the use of the repair. Burton's addition to the repair has added a definitely superior wall (using external oblique aponeurosis) to an unyielding attachment (Cooper's ligament). It is left to the discretion of the operator to choose the wall which best suits the need.

A female patient, age 52 yrs., was admitted to the Boston City Hospital for cancer of the transverse colon, but precipitated a large femoral hernia, which became incarcerated and was rapidly becoming strangulated. It was reduced at once and operated upon the next day. Figure 4 shows the large 6 cm. hernia sac,

a relatively adequate transversalis fascia, a weak Poupart's ligament, common in females, and Cooper's ligament, which is situated more superficially in the female due to the flatter, broader pelvis. Figure 5 shows the silk sutures placed between Cooper's ligament and the transversalis fascia and the conjoined tendon. The external oblique aponeurosis is closed and then the skin is sutured. The patient's condition would have permitted a more radical repair, but it was not necessary in the opinion of the surgeon. The cancer of the colon was successfully operated on two weeks later, and the repair, upon abdominal examination, was found to be totally adequate. It was particularly so in the region about the vascular outlet.

Our experience at the Boston City Hospital from 1940 to 1948 falls into line with most of the recognized principles of hernia study, but there are several factors that will be stressed. In general, the incidence of femoral hernia seems to be increasing and the geriatric implication of the disease is becoming more evident. Table I shows that the average age of the 174 cases was 60 yrs. When these statistics were broken down, it was found that of the 146 cases that survived operation, the average age was 57½ yrs., and of the 28 that died, the average was

TABLE III

Operative Deaths—24 Cases—14%

| | | |
|---|---|---|
| *Sac Content* | | |
| 1. Bowel | 23 | (gangrene—10) |
| 2. Omentum (only) | 1 | |
| 3. Incarcerated | 4 | (resected—14) |
| 4. Strangulated | 19 | (anastomosis) |
| *Complications* | | |
| 1. Heart Failure | 11 | |
| 2. Intestinal Obs. | 7 | (Peritonitis—2) |
| 3. Pneumonia | 5 | |
| 4. Arteriosclerosis | 5 | |
| 5. Anemia | 3 | |
| 6. Cancer of Colon | 1 | |
| 7. Uremia | 1 | |

73½ yrs.—a difference of 16 yrs. Femoral hernia, as the statistics bear out, is a disease of the aged and with the prolongation of the life span, it will become a more frequent occurring condition for the same reasons that cancer, heart disease and degenerative diseases are increasing. The fact that elderly people do not stand operation well is re-emphasized and that elderly patients allow complications to arise before seeking relief has also been noted.

The preponderance of female over male incidence is evident but it is not as overwhelmingly partial in our group of patients as is generally taught. The ratio has been quoted to vary from 1 to 2 or 1 to 3 in favor of the female. There is no apparent reason why our sex incidence should more nearly approach an even distribution. Likewise, we offer no suitable answer for the prevalence of the right sided hernia unless one assumes that undo strains result from right handedness or right footedness. In all probability, the incidence of bilateral herniation, or at least the tendency toward bilateral occurrence, is more frequent than we have found. In the case of the female patient which was presented, a small weakness was found on the right side at the subsequent operation but which was not evident by manual examination externally. Theoretically, she could have developed a

hernia on this side if she had been exposed to additional stress; or, she may still develop one on that side at some later time.

The surgical approach was evenly distributed between the suprainguinal and and the infrainguinal routes. This doesn't give the true picture of the trend. During the first few years of the study, almost all of the surgeons approached the hernia from below Poupart's ligament. Toward the end of the study, most of the operations have been performed from above. In all probability this is due to a better understanding of the anatomy of the region; and it is particularly due to the recognition of the versatility and safety in the use of this approach. Since so many types of operation are available to the surgeon and he may be wholly unfamiliar with the ones he himself does not use, we find that we cannot be too

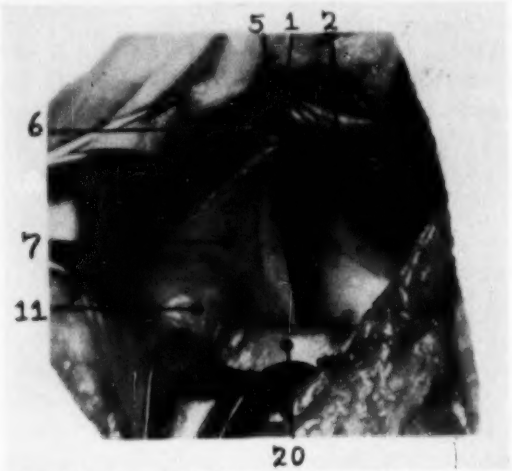


Fig. 4—(Case presented)—Left femoral hernia in a female, age 52 yrs., showing transversalis fascia, a thin Poupart's ligament, both retracted. Cooper's ligament exposed. Reduced femoral hernia shown. **1**-Cooper's ligament; **2**-upper margin of incised transversalis fascia retracted to expose more of Cooper's ligament; **5**-region of pubic spine; **6**-Poupart's ligament; **7**-inferior portion of incised transversalis fascia overlying Gimbernat's ligament, to which it is adherent; **11**-weakness in ligamentous structure of the trihedral space of Burton.

dogmatic about the recommendation of any one procedure over another. The principle of a surgeon using an inferior method well, might be better than one using a superior method badly. The situation is similar to the different battery of tests that various internists use to arrive at the same conclusion in liver disease. It is imperative then, that we know our procedures well and to use them in their suitable situations. Many times in attempting a repair, the surgeon will pull and stretch weak tissues until they become shredded so that they rupture at the time, or break down at some later date. He must not underestimate the strength and adequacy of the tissue used in the repair. This point may be quite difficult to be certain about in some cases at the time of operation and even the best will falter on occasion. Most recurrences will be found in this group. Experience will probably dictate that we be more careful in these doubtful cases.

In 8 of the cases, a previous herniorrhaphy was performed in the inguinal region of the same side in which the femoral sac either developed later or was present at the time of operation and went unnoticed. These cases clearly emphasize the advisability of looking for multi-loculated sacs in the inguinofemoral region. It is not likely that these hernias developed as so-called "recurrences" following an inguinal repair but that they are altogether new hernias developing because of an inherent weakness in the femoral region either before or after operation.

The gravity of the hernia situation in the femoral canal is shown by the number of strangulations and incarcerations. In analyzing the statistics, if a hernia was incarcerated as well as strangulated, it was classified as incarcerated; whereas a strangulation alone was classified as such. Almost two-thirds of the cases showed either one or both of these complications. Most people have a tendency to let a femoral hernia "ride" until it causes some trouble. This may be due to the fear that the older age group holds for hospitals in general and operations in particular. This type of hernia also lacks a "fixation point", for a lump on the leg doesn't mean as much to them as a mass in the scrotum and they refuse to recognize its

TABLE IV

CAUSES OF DEATH ON 4 UNOPERATED CASES

1. Pneumonia—Nephritis—Uremia
2. Pneumonia—Empyema
3. Pneumonia—Cerebral Thrombosis
4. Heart Failure—Strangulated Hernia

TABLE V

FOLLOW-UP CLINIC ON 53 CASES
AUGUST 1949

| | 1-9 years | |
|---|---|---|
| Pain | 6 | Recurrent 9.5% |
| Swelling | 3 | |
| Recurrence | 5 | |

danger. Operating under these conditions showed a relatively high mortality of 14 per cent in spite of the institution of measures to combat dehydration, electrolyte imbalance, anemia, hypoproteinemia, septicemia. Of the four cases that died unoperated upon, 3 succumbed to pulmonary infection and 1 to cardiac failure.

Only 53 cases of the 146 cases that survived operation, answered the query for follow-up studies. Many had moved to unknown addresses and a number had died of intercurrent diseases and other complications. Six had pain which varied from aggravating to mild. Four localized it in the incision and two situated it deeper within. In none did it cause any incapacity. Three had swelling at the site of operation which was a vague complaint and not noted at the time that the patient was examined. The recurrence rate of 9.5 per cent (5 cases) is comparable to the reportable incidence of 5-22.5 per cent.

## SUMMARY AND CONCLUSIONS

1. Femoral herniae in a large charitable hospital (a) will occur at an average age of 60 years, (b) will have a survival age averaging 57½ years, and (c) the average age of the death cases will be 73½ yrs. Almost all of the death cases will

have an incarcerated or strangulated hernia in which the bowel is gangrenous, requiring resection and anastomosis of the bowel. Other serious complications, like heart failure and pneumonia are present on admission, causing the patient to be moribund and the case to be inoperable.

2. The surgeon will be limited in his choice of repair by the age of the patients and the complicating diseases, but he should always attempt to perform an adequate repair.

3. The operative approach above Poupart's ligament offers a better access to the neck of the sac, inspection of its contents, resection of gangrenous bowel if necessary, and permits the use of the strong Cooper's ligament as the inferior

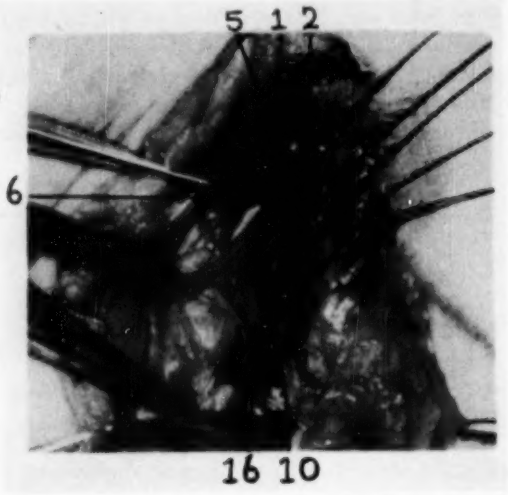


Fig. 5—(Case presented)—Silk sutures placed between the conjoined tendon and transversalis fascia, and Cooper's ligament. **1**-Cooper's ligament; **2**-transversalis fascia, upper leaf; **5**-region of pubic spine; **6**-thin Poupart's ligament; **10**-femoral vein; **16**-femoral ring.

and posterior leaf of the repair in femoral hernias. An abdominal wall that will repel intraabdominal pressure, must be used to suture to Cooper's ligament.

4. The operative approach above Poupart's ligament permits the surgeon to inspect the posterior inguinal wall for weakness, deficiences, or other locules and their correction by the operation of choice.

5. The interfering epigastric and the obturator vessels should be ligated so that the line of repair can be made without interfering with its continuity.

6. We recommend the use of a more permanent type of suture than the absorbable sutures.

We want to thank Leo Goodman of the Mallory Institute of Pathology, of the Boston City Hospital, for the excellent photographic work.

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# WATER-SOLUBLE CHLOROPHYLL IN THE TREATMENT OF PEPTIC ULCERS OF LONG DURATION*

## A Preliminary Report

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The broad problem of the treatment of peptic ulcers involves the discussion of complex etiological factors which may vary with each specific case. Yet at the root of the problem is always the ultimate requirement—to bring about healing of the ulcer crater. Whereas a specific therapy for accelerating the healing process will not in itself obviate the necessity for careful consideration of divers factors which will continue to complicate the overall treatment of peptic ulcers, such a therapy can play an important role in the reparative stage of almost all peptic ulcer cases. It is from this point of view that this clinical study is presented.

The accepted manner of bringing about the healing process has been to create an environment at the site of the affected area that will allow the natural regenerative processes of the gastroduodenal tissues to regain the upper hand. The erosive action of hydrochloric acid is combatted by antacids and/or the ulcer crater is protected from the aggression of the gastric juices by a coating of some mucilaginous material; special diets are instituted to minimize the secretion of acids and there is a determined effort to eliminate psychological factors.

Such therapies, and combinations thereof, have proven of obvious value in helping to create the environment necessary for eventual healing while controlling the subjective symptoms of peptic ulcer. Yet even where this preliminary success is obtained, regenerative properties of the tissues may remain weak and healing proceed slowly if at all. The high incidence of patients who have gone through one ulcer regimen after another with temporary relief, but with no healing of the ulcer crater suggests the need of supplying direct assistance to the damaged gastroduodenal tissues in the form of a tissue-stimulant.

Water-soluble chlorophyll† has been shown in numerous clinical and laboratory reports to possess tissue-stimulating properties. Boehme[1] reported on the use of chlorophyll at the Lahey Clinic in more than 50 cases of chronic leg ulcers with a high percentage of rapid healing. Many of these cases were unhealed from one to eight years but obtained complete healing by chlorophyll therapy in from six to ten weeks. Smith and Livingston[2, 3] produced experimental surgical wounds in 1,372 animals, and found that chlorophyll reduced the healing period by 25 per cent in more than two-thirds of the cases in comparison with controls. The cell-stimulating properties of chlorophyll reported by these clinicians has been substantiated by many subsequent reports on the treatment with water-soluble chlorophyll of acute and chronic wounds, burns and dermatoses[8, 9, 10].

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*Read by title before the Fourteenth Annual Convention of the National Gastroenterological Association, Boston, Mass., 24, 25, 26 October 1949.

†Whenever the term chlorophyll is used, the water-soluble derivatives, "Chloresium" grade, are meant, the term chlorophyll being used solely for the sake of brevity.

It was thought that if chlorophyll could be brought into contact with the site of the lesion, accelerated healing of the ulcer crater might result. It would be desirable to keep the chlorophyll in direct contact with the ulcer over an extended period of time in order to afford the chlorophyll ample opportunity to exert any effect it might have in this environment.

Through the cooperation of Rystan Company, Inc., a chlorophyll powder preparation was obtained which, in addition to maintaining the chlorophyll in contact with the site of the lesion would also provide a protective coating for the lesion, and antacids to neutralize excessive gastric acidity. The antacids used were aluminum hydroxide and magnesium trisilicate, but it is our opinion that any antacid may be used in place of these, so long as it does not cause any change in the acid-base equilibrium of the blood (e.g. alkalosis).

It was felt that with this preparation it would be possible to provide the same desirable functions obtained from currently used ulcer preparations through the action of the antacids and the protective coating for the lesions. The addition of the water-soluble chlorophyll might be anticipated to *accelerate the healing process where healing might occur in due course in any case, and promote healing where lowered tissue resistance might otherwise result in a static condition.* The clinical series described below was designed as a means of determining whether there was potential value in the chlorophyll ingredient. More precise determination of the nature of the contribution of the chlorophyll ingredient will require more extensive and strictly controlled studies.

In this series of cases, we made important deviations from the orthodox ulcer regimens. Ordinarily in the treatment of peptic ulcer, strict diet, restriction of smoking and alcoholic beverages, and sometimes restricted activity, is prescribed. No special diets were prescribed in this series, although it was stipulated that there should be no food taken between meals. There was no program of rest, and patients were instructed to pursue their normal habits as regards smoking and the taking of alcoholic beverages. It was felt that the avoidance of this part of the normal ulcer regimen would eliminate some of the factors whose beneficial effects are ordinarily difficult to disassociate from the effects of the preparation under clinical study. Thus, in this series the results should provide a basis for a direct evaluation of the chlorophyll powder preparation.

In the selection of patients for this series, positive evidence of the presence of ulcers was obtained by x-ray in every case. The patient was instructed to take one of the powders on arising, a powder one-and-a-half hours after breakfast, one after lunch, dinner and before retiring. Instructions were to place the powder on the tongue and wash it down with a small amount of water. An alternative was to place the powder in a tablespoon with some water and then swallow immediately. On completion of treatment, patients were x-rayed and reports were submitted by the roentgenologist. The same roentgenologist made all before and after diagnoses.

The chart below gives a summary of the cases completed in the first of two clinical series. It will be noted that all of these cases had long ulcer histories

previous to the institution of treatment with the chlorophyll preparation. The majority of these cases had undergone treatment with accepted therapies incorporating strict diets, antacids, aluminum gels, and mucin preparations.

In 20 out of the 27 cases in this series symptomatic relief was striking. Pain subsided and other subjective symptoms disappeared with startling regularity 24 to 72 hours after treatment was started. Those who had complained of night pains were able to sleep soundly without taking sedatives or narcotics. Of the remaining seven cases, four obtained moderate symptomatic relief and three showed no improvement.

No toxicity was encountered, nor was there any tendency towards constipation or diarrhea. If anything, there was a tendency to promote regularity. It was observed that patients who had suffered weight loss due to previous restricting diets gained back this weight under the chlorophyll regimen.

There were three cases of duodenal ulcer with pyloric obstruction which were included in this series although there was little reason to expect a favorable response. After three weeks of treatment showed negligible progress, these patients were sent to surgery. X-ray examination after treatment was omitted in these cases.

From the table it may be noted that of the 24 patients examined roentgenologically after treatment, 20 showed x-ray evidence of complete healing after from two to seven weeks treatment. Of the four who showed no roentgenological improvement, two had evidenced great clinical improvement. "X-ray Results" are as reported by the roentgenologist. This series of 27 cases ran from March, 1949 to October, 1949. Through January of 1950 there have been no recurrences in the 20 cases which showed complete healing.

There were several cases in this series in which the clinical experience was particularly interesting:

*Case 2*:—A 53 year old white male was admitted to the hospital in a state of shock. Stools were black and tarry and peptic bleeding was evident. He could furnish no background beyond the fact that some 25 years previously he had undergone a gastroenterostomy. When bleeding ceased, a gastrointestinal series was run to determine the extent of the pathology with the following report from the roentgenologist. "Severe spastic deformity at the site of the gastrojejunostomy. Pocketing also present. Emptying of barium through the stoma somewhat delayed."

The patient was placed on the chlorophyll regimen with no restrictions as to diet. Sixteen days later, x-ray showed the spastic deformity at the site of the gastrojejunostomy to have disappeared. The pocket previously seen was absent and emptying of barium greatly improved. The roentgenologist reported, "Healed marginal ulcer with improved gastric emptying at the site of the gastrojejunostomy."

*Case 10*:—A 43 year old male who, for the past six years, had suffered with severe heartburn, which came on after meals and was relieved by milk. For the

TABLE I

| Case | Age | Sex | X-ray Diagnosis | Dur. of Previous Treatment | Length of Chlorophyll Treatment | Clinical Results | X-ray Diagnosis After Chlorophyll Treatment |
|---|---|---|---|---|---|---|---|
| 1 | 31 | F | Gastric | 2½ years | 2 weeks | Greatly improved | Healed ulcer |
| 2 | 53 | M | Marginal | Not known | 2½ weeks | Symptomless | Healed ulcer |
| 3 | 46 | F | Duodenal | 10 years | 4 weeks | Greatly improved | Healed ulcer crater |
| 4 | 25 | M | Prepyloric | 3 years | 4 weeks | Greatly improved | Healed |
| 5 | 44 | M | Duodenal | 2 years | 4 weeks | Greatly improved | Healing duodenal ulcer |
| 6 | 41 | M | Duodenal | 6 years | 4½ weeks | Greatly improved | Healed ulcer |
| 7 | 38 | F | Duodenal | 15 years | 6 weeks | Greatly improved | Showed no change |
| 8 | 55 | M | Duodenal (Pylorospasm) | 2½ years | 3 weeks | Greatly improved | Healing duodenal ulcer |
| 9 | 42 | M | Duodenal | 12 years | 7 weeks | Greatly improved | Healed ulcer crater |
| 10 | 43 | M | Duodenal | 6 years | 5 weeks | Greatly improved | Healed ulcer |
| 11 | 45 | M | Duodenal | 5-6 years | 5 weeks | Greatly improved | Ulcer crater gone. Scarred bulb remains. |
| 12 | 40 | F | Duodenal | 2 years | 4 weeks | No improvement | No improvement or change |
| 13 | 48 | M | Postbulbar | 9 years | 6 weeks | Occas. Pain Per. | Healed postbulbar ulcer |
| 14 | 53 | M | Duodenal | Not known | 8 weeks | Slow improvement | No improvement |
| 15 | 52 | F | Penetrating duodenal | 12 years | 4 weeks | Greatly improved | Healed ulcer |

TABLE I (continued)

| Case | Age | Sex | X-ray Diagnosis | Dur. of Previous Treatment | Length of Chlorophyll Treatment | Clinical Results | X-ray Diagnosis After Chlorophyll Treatment |
|---|---|---|---|---|---|---|---|
| 16 | 53 | F | Duodenal | 6 years | 6 weeks | Greatly improved | Healed ulcer |
| 17 | 37 | F | Duodenal | 4 years | 6½ weeks | Greatly improved | Healed |
| 18 | 30 | M | Duodenal | 7 years | 5 weeks | Greatly improved | No improvement |
| 19 | 56 | M | Duodenal | 6 years | 3 weeks | Greatly improved | Healed |
| 20 | 28 | M | Duodenal | 12 years | 3 weeks | Greatly improved | Healed |
| 21 | 38 | M | Duodenal | 12 years | 3 weeks | Greatly improved | Markedly improved |
| 22 | 47 | F | Duodenal | 2 years | 3 weeks | Greatly improved | Healed ulcer |
| 23 | 60 | M | Duodenal with pyloric obstruction | Not known | 3 weeks | Moderate improvement | Omitted |
| 24 | 62 | M | Duodenal with pyloric obstruction | 40 years | 3 weeks | No improvement | Omitted |
| 25 | 47 | M | Duodenal | 6 years | 3 weeks | Moderate improvement | Healed |
| 26 | 34 | F | Duodenal with pyloric obstruction | Not known | 3 weeks | No improvement | Omitted |
| 27 | 56 | M | Duodenal | 7 years | 3 weeks | Greatly improved | Markedly improved |

Above studies reported on by Morris Slobodkin, M.D. (NACR) Roentgenologist
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past two weeks epigastric pain had been very intense and various antacids had afforded no relief. Roentgenograms taken a few days previously, which he brought with him, revealed a large duodenal ulcer.

This patient was leaving on a cross-country trip, so without any great optimism a month's supply of the chlorophyll powders was given to him with instructions to report back on his return. His information, when he did return one month later, was that he had been able to eat anything and everything, smoke as much as he chose and had felt fine the whole time. The aggravations incident to traveling had apparently not bothered him at all and he was inclined to doubt that he had actually had an ulcer in the first place. An x-ray study at this point showed the duodenal ulcer, previously noted, to be completely gone.

The striking element in this case is that all the items normally avoided in a controlled routine of treatment were indulged in by this patient with no apparent detrimental effect. Rough and spicy foods, tobacco, and psychogenic unrest had not affected his wellbeing. His pain and distress had disappeared with the third powder.

*Case 15*:—A markedly different condition was exemplified in this case. This was a 52 year old white woman whom I had treated at varying intervals during the past three years. Her ulcer story dated back 12 years. She had been on ulcer diets, had used various antacids, aluminum gels, mucin preparations, and when pain was unbearable had used narcotics. She was in excruciating pain and x-ray studies led to a diagnosis of a penetrating duodenal ulcer. A constriction of the mid-portion of the duodenal bulb was also noted.

She was placed on the chlorophyll powders and obtained immediate relief. Her diet was not restricted and her smoking amounted to approximately half a pack of cigarettes daily. At the end of four weeks x-ray studies showed the duodenal ulcer to have disappeared and the degree of mid-bulbar spasm to have diminished. In this case the prompt subsidence of pain and the disappearance of the ulcer crater were felt to be quite remarkable.

*Case 17*:—A 37 year old white female whose knowledge of the presence of an ulcer dated back about four years. She had followed various diets and tried many medications. Antacids afforded moderate relief as did milk or sodium-bicarbonate. She described pressing sensations in her epigastrium accompanied by heartburn, and was often kept from sleep by the pain. A gastrointestinal study showed a persistently deformed and contracted duodenal bulb.

After instituting the chlorophyll regimen, pain disappeared, although slight nausea persisted for three days. By the end of one week the patient was able to eat whatever she chose without experiencing nausea, heartburn or pain at night. A follow-up series of x-rays taken at the end of six and one-half weeks showed the duodenal bulb to be neither contracted nor deformed.

Here again the prompt subsidence of pain was striking and the x-ray evidence of improvement very marked.

*Case 13*:—One other case is worthy of particular mention. This patient was a 48 year old white male whose ulcer history dated back nine years. A gastro-

intestinal study at that time had revealed a peptic ulcer. Through the years he had suffered from heartburn, nausea and night pain. He had found temporary relief in the aluminum gel preparations.

X-rays taken at the beginning of the chlorophyll treatment showed a narrowing and deformity of the descending duodenum and the presence of an ulcer measuring 8 mm. in length.

After three days of chlorophyll therapy, his symptoms subsided with only occasional epigastric discomfort remaining. X-ray taken at the end of six weeks showed that although a narrowing of the horizontal duodenal loop persisted, there was no longer any sign of the presence of an ulcer crater. This was reported by the roentgenologist as a healed postbulbar ulcer of the duodenum with residual deformity. The case serves to again emphasize the control of pain and the healing effect that the chlorophyll preparation seems to provide.

The encouraging results obtained in the above series of cases prompted an extension of the trial of the chlorophyll powder preparation. Therefore from October 1949 through January 1950 the use of this preparation was continued and an additional 52 cases were treated. A substantial addition to the original series of 27 cases was obtained. The results in this series of 52 cases paralleled those of the smaller original series very closely.

Of the 52 cases, two had pyloric obstructions and did not respond to chlorophyll therapy. These were referred to surgery without final x-ray.

Of the remaining 50, 38 patients showed complete healing on x-ray examination and were symptom free. Two more patients experienced complete and lasting symptomatic relief, although x-ray examination failed to show the ulcer crater to have disappeared.

The outcome in the remaining ten cases was not considered to have been favorable. In these cases symptomatic relief ranged from slight to moderate and healing of the ulcer crater was not evident on x-ray examination. The chlorophyll regimen in these cases did not improve on previous therapy employed.

## Summary

A total of 79 patients with x-ray proved duodenal and gastric ulcers were treated with a powder incorporating water-soluble chlorophyll, a "coating" material, and recognized antacids. The treatment was not accompanied by the usual restrictions on diet, smoking, alcoholic beverages, or daily activity.

Five patients were diagnosed as having duodenal ulcer with pyloric obstruction; these cases, as would be expected, showed no improvement through use of the chlorophyll preparation, and roentgenological examination after treatment was omitted.

*Healing of ulcer crater*:—Of the remaining 74 patients, 58 showed, on roentgenological examination, complete healing in from two to seven weeks; 16 patients showed no roentgenological improvement, but of these, two experienced great clinical improvement and two were symptom free.

*Symptomatic relief*:—Of the 74 patients, 60 experienced complete symptomatic relief in from one to three days. Two had moderate relief. The remaining 12 were not benefited.

No patient experienced any toxic reaction from the chlorophyll powders. There was no tendency towards constipation or diarrhea.

There were no recurrences of symptoms in 20 of the cases showing complete healing, which were followed for periods of from four to 11 months.

## Comment

Insufficient time has elapsed to evaluate the recurrence factor, yet the immediate results obtained with the chlorophyll powder in the treatment of peptic ulcers of long standing are impressive.

The fact that the usual restrictions on diet, activity, smoking and alcoholic beverages were not incorporated in this series indicates that the successful clinical and roentgenological results obtained can be directly attributed to the chlorophyll powder treatment. That these restrictions can safely be omitted in the treatment simplifies the physician's task of insuring patient cooperation. However, the possibility is also suggested that other researchers may obtain even better results with this preparation by including these known beneficial measures in their regimen of treatment.

There was reason to believe that the chlorophyll preparation might exert a healing effect. The actual clinical results suggest, however, that the chlorophyll ingredient may have provided therapeutic effects in addition to the tissue-stimulating properties. The prompt and lasting symptomatic relief, for example, suggests some contribution on the part of the chlorophyll similar to the analgesic effect noted by Cady[4], Morgan [4, 5] and others in the treatment of external lesions.

Recently work by Rapp and Gurney[6] at Loyola University established that water-soluble chlorophyll inhibits the action of proteolytic bacteria and enzymes. It is not inconceivable that chlorophyll action against proteolytic agents in the intestinal tract may play an important role in bringing about the favorable results experienced. This conjecture suggests further a connection with the lysozyme activity associated with lesions of the intestinal tract recently reported by the Columbia group[7].

Rapp and Gurney[6] also demonstrated the ability of water-soluble chlorophyll to raise the pH in human saliva. Our studies make no attempt to evaluate such an effect in ulcer therapy, but this factor too seems worthy of investigation.

Continuation and expansion of these studies seems definitely indicated. On the basis of results to date, it is my opinion that water-soluble chlorophyll should have an important part in the medical management of peptic ulcers.

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# CORTICODIENCEPHALIC GASTROINTESTINAL SYNDROMES IN EPILEPTICS*

## (PART V)

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### CEREBRAL CORTEX

Now that we have presented the literature on the nuclear connections and constituents of the anterior nucleus of the thalamus and have definitely traced its fibre connections to the cerebrum, we will trace the fibre connections of the areas within the cerebral cortex to which the anterior nucleus of the thalamus sends projections. In this way the mammillothalamic tract will be definitely established as deeply rooted within the cerebrum as a corticothalamomammillomesencephalic tract. It will furthermore be shown that the cortical portion of the corticothalamomammillary fibre system is not merely connected in one but in many places with the cerebral cortex and can possibly be traced to all lobes. The thalamocortical fibres from the anterior nucleus of the thalamus have been shown above to be connected with the precentral area. The work of Watts and Fulton has shown how the neurogenic control by the cortex in the region of the sigmoid gyrus of the gastrointestinal tract is established. Not only the sigmoid gyrus but various motor and prefrontal regions function on an autonomic basis. It is therefore not going far afield to conclude that the fibres going from the anterior nucleus of the thalamus are deeply rooted in various areas particularly the zones situated in the frontal lobe. That these cortical areas within the frontal lobe which are connected with the mammillothalamic contain various other functions particularly those of chewing, tongue action, and even eye movement will be seen from the following presentation of the literature. Our seemingly irrelevant accentuations of the work of Davison and Goodhart on cranial nerve action in the head and eye movements had a distinct purpose. All such functions have been firmly established by Muskens and others as connected with the parasympathetic chain and the medial longitudinal fasciculus. Therefore this anatomic scheme is nothing but a tract for the neurogenic control of gastrointestinal and other autonomic activities. Eye movements will be included in our dealing with the frontal lobe in relation to the mammillothalamic tract in order to strengthen our presentation of the literature by Davison and Goodhart when we previously dealt with this subject. We will, however, concentrate on the specific portions of the frontal more than do

*Read in part before the Thirteenth Annual Convention of the National Gastroenterological Association, New York, N. Y., 7, 8, 9, 10 June 1948.
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Davison and Goodhart due to its connection with the mammillothalamic system. Kappers, Huber and Crosby state that in man the ventral portion of the stimulable cortical area does not extend in front of the border of the sulcus precentralis agranularis, but the dorsal portion of such areas, and those concerned in eye movement appear to be in the second frontal convolution (Mills 1904), (Sahli 1905). The areas in this region are the area precentralis agranularis (area #6 of Brodmann) and the intermedial precentral cortex of Campbell (1903, 1904, 1905). The functions in these areas are parasympathetic.

In the following detailed description of the frontal lobe we will cover not only the centers for eye movement, but also those involved in the action of the facial and laryngeal musculature. The functions of the frontal lobe are intimately connected with those of deglutition and mastication. Any item which deals with

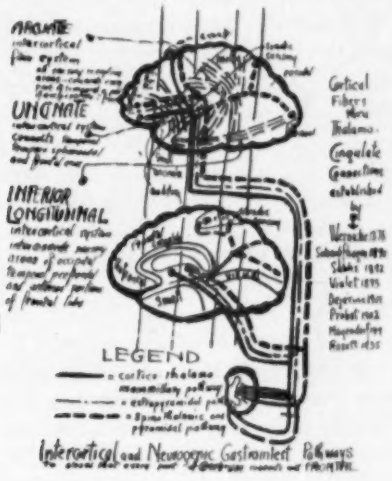


Fig. 27

lower cranial nerve function is essentially autonomic and hence neurogenically gastrointestinal. That the above is true has been shown by the great many references to the medial longitudinal fasciculus. Such functions form an integral part of the mammillary region which continues into it. If we have failed to impress the reader with this important fact in tegmental neurology of the mammillothalamic system it was because the title of our paper calls for confinement to the cerebrum. In a very lengthy elaboration of the functions, particularly those of the lower forms, rhythmic chewing is intimately associated with gastrointestinal functions in the frontal lobe. With the presentation of the areas of the frontal lobe which are connected with the functions of this group of muscles as well as the eye movements we will give specific areas of established cytoarchitectonic value. In this way the reader will become familiar with those structures in the frontal lobe which are intimately connected with the terminations of the mam-

millothalamic tract. Kappers, Huber and Crosby claimed that stimulation of this region (area 6a), as well as the pathologic condition involving it produced conjugate lateral movements of the eye and head. This is connected with all motor cells which lie within the regio precentralis gigantopyramidalis. Attention is drawn particularly to the center for the muscles of the tongue, the larynx and the lips, the motor stimulable areas for which lie caudal to the subregio frontalis inferior, in close relation to Broca's center for articulate speech but not within it (Krause 1904). From the above one can see what a wide range of interconnective functions are found in the areas stimulated by Watts and Fulton (sigmoid gyrus), and the cingulate terminations of the mammillothalamic tract. The region for rhythmic chewing in the lower forms extends further than the precentral region. This is likewise true for the areas dealing with eye movements which was shown in carnivores to be situated in front by Ziehen (1899), Fleishmann (1906), Chapman (1906), and C. & O. Vogt (1907). The last mentioned authority tends to place this in the lower part of the precentral gyrus (pyramidal system).

Although the optic thalamus has the function of emotional expression and the fibres from this structure stretch into the cerebral cortex, particularly into the frontal region and have been assigned to the transmission of such impulses directly into the frontal cortex, we will not at this time, elaborate upon such functions. That these same structures subserve the functions of the gastrointestinal tract is merely incidental. This elaboration should be constantly kept in mind and connected with the areas which Watts and Fulton repeatedly emphasized. It is in such and nearby areas in which we find the fibres of the mammillothalamic region. One should therefore automatically connect chewing, gastrointestinal motion, etc. with other functions that have been assigned to the mammillothalamic system such as sham rage, manifestations, and other evidence of musculoskeletal activities associated with the emotions and intimately bound (Cannon) gastrointestinal function. Although such manifestations have been produced by stimulation of the lower portions of the diencephalon nevertheless it hasn't been extensively elaborated upon in the literature that the functions are located within the fibres that terminate in the upper portions of the mammillothalamic tract. For details of the anatomy of the above and the comparative neuroanatomy of the areas concerned with eye movements, we refer the reader to Kappers, Huber and Crosby. These authors state that the forehead and eye movements extend farther frontalward in the lower forms such as in macacus (Beevor and Horsley 1894). The center for synchronized movements of the head is situated in the region of the ramus dorsalis of the sulcus cinguli while in semnopithecus C. & O. Vogt located it directly above this branch. In the chimpanzee, Grünbaum and Sherrington (1906), located it in the region of the ramus lateralis of the sulcus frontalis. These facts indicate not only the clearly frontal position of these functions of the motor area, but also call attention to the real value of the morphologic relationship existing between the ramus dorsalis arcuatus and the sulcus frontalis superior (compare also Cole 1911). Cytoarchitectonic features of certain areas of this region are very important because they are related to cranial nerve and

gastrointestinal functions. The work of Foerster and Penfield has been related to pupillary changes by virtue of head movement and other cranial nerve action. This association of sympathetic with parasympathetic action has been well established experimentally and permanently stamped by the work of Brodmann and others who are primarily microscopic anatomists. Stimulation of certain areas of the brain by Penfield has evoked certain special reaction for definite areas. These reactions are said to be related to the cellular structure of certain areas as described. Area #6a, b has been a common place of stimulation by Penfield. This area has likewise been assigned various pyramidal as well as extrapyramidal function as seen in Figure 25. Watts and Fulton assigned gastrointestinal motor

Fig. 28

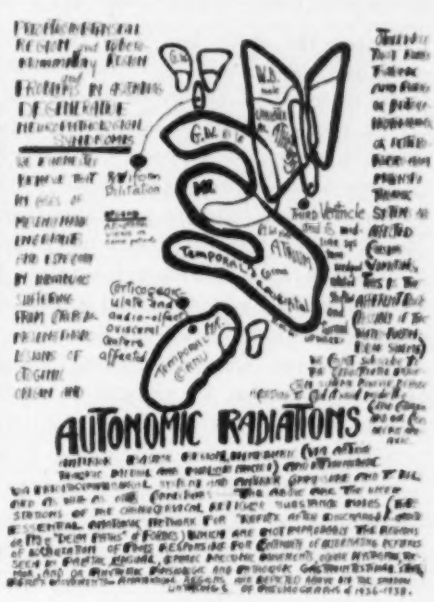


Fig. 29

function to some areas. Penfield, and Davison and Goodhart, as well as others located extrapyramidal function, eye movements, head movements and chewing movements in these areas. On the basis of the above work, but mostly from his own anatomic experiments, Muskens went further and connected such areas and extrapyramidal functions with his medial and posterior longitudinal fasciculus system. In doing so he established a parasympathetic cranial nerve chain which carries gastrointestinal neurogenic cells. The area gigantopyramidalis, because of its relation to these other areas, is a very important one. A number of cells (large pyramidal cells of Betz), estimated at about 80,000 for man and 13,000 for the chimpanzee (Campbell 1905), will run parallel with the number of pyramidal fibres. In the ape and in man a large part of the regio precentral agranularis

(area 6) forms area gigantopyramidalis (Brodmann 1906 and 1909), only through the absence of the giant pyramidal cells. The functional differences between this and the area gigantopyramidalis (area 4 of Brodmann) according to the analysis of Mills (1901), and particularly that of Wilson appeared to consist in the fact (Kappers, Huber and Crosby) that the area gigantopyramidalis has a direct motor effect while the area precentralis agranularis of Brodmann (area 6), the intermedial precentral cortex of Campbell (1903, 1904 and 1905), and the midfrontal cortex of Mills has a tonic function. This tonic function for pyramidal and extrapyramidal cortical areas includes also gastrointestinal tonus. That this is probable has been shown previously when fibres from the anterior nucleus of the thalamus were traced to the motor cortex.

From the above data, which we incidentally started in relation to eye movement and the mimetic expression, chewing, and other functions, we have eventually traced the functions of motion and tone, both of which are intimately connected not only with musculoskeletal but also with gastrointestinal and other divisions of the autonomic system. The cortical representation of the mammillothalamic tract is therefore intimately bound with not only the musculoskeletal functions which subserve gastrointestinal activity, activity of the emotions, but also head, eye, neck, and body movement involved in the epileptic attack. Functions which have therefore heretofore been assigned as being limited to the diencephalon are seen to extend in that part of the mammillothalamic tract which ends in the frontal cerebral cortex. Here we again remind the reader that by using the term "mammillothalamic" we do not choose to exclude the other diencephalic tracts that are situated at the base, i.e. the fibres of the preoptic components of the striae terminalis, the dorsal longitudinal fasciculus and any connections with the cortex from the Friedemann-Cajal loop containing Gansers commissure and other structures favored by the experimentalists on gastrointestinal reactions to hypothalamic stimulation and by the extensive array of famous investigators of the subject of water metabolism. We also see from the above that our extensive elaboration and references to Muskens on the subject of tonus in relation to myoclonic shocks has also not gone far afield since movement as well as its abnormal type of reaction in the form of variations in tonus is located in this region of the frontal lobe. The exact location of this tonus has been shown by Kappers, Huber and Crosby to be in the regio precentralis agranularis (area 6 of Brodmann), the intermedial precentral cortex of Campbell and the midfrontal cortex of Mills. Although the above areas have been shown to have tonic functions, (the last mentioned region has a direct motor center such as that for conjugate eye movement), there are other centers, among which are those in which the tonic character is very clear, as for example in the center of functional expression of joy and anger (corrugators of the eyebrow, and the muscles of the orbicularis oculi and of the wing of the nose). Mills and Frazier (1905), also mention with the above the visceral center, the role of which is so evident in emotion such as the center for tear secretion, perspiration and similar centers.

Now that we have elaborated upon the various centers of the cerebral cortex which form the upper end of the mammillothalamic tract and have shown that not only is the sigmoid gyrus and its adjacent region connected with the area which has the neurogenic control of gastrointestinal functions, but also that the cingulate gyrus has been repeatedly referred to as being intimately connected with this great nucleofascicular system, the reader will understand that some specific cortical pathway has to be chosen to interconnect all the related gastrointestinal and autonomic functions and attach them to the mammillothalamic and other diencephalic tracts in which gastrointestinal neurogenic control is experimentally proved.

We have taken the space above for the presentation of the literature dealing with certain specific areas in the frontal region with which the mammillothalamic tract comes in contact, not only for the purpose of showing how elaborate a network of functions the frontal region contains but also that this region is important in a number of ways. It contains the centers of the neurogenic control of gastrointestinal and urinary function not only by its direct relation to the upper end of the mammillothalamic tract and thus leading into the hypothalamus and related centers, but also because it is connected with the area gigantopyramidalis. This last area is commonly known to be connected with the pyramidal pathways. Due to the above, a telencephalic pathway must therefore be chosen to connect the frontal with other areas. The reader will recall that we have previously divided the schools on the neurogenic control of the gastrointestinal functions into two groups namely the mammillothalamic group and the pyramidal. We have previously aligned (as taken from the literature) the various exponents of these schools. The reader has been referred to the papers by Hare, Sheehan, Watts and Fulton and to the experiments of Muskens.

Later we showed how it is not improbable for some neurogenic functions of the gastrointestinal system to be localized in other portions of the cerebral cortex than the frontal. Spiegel's centrifugal and centripetal pathways carrying control of gastrointestinal function were mentioned. The postcentral, parietal, temporal and other parts of the cerebral hemisphere than the frontal were shown to carry gastrointestinal function. We must therefore show how these areas could possibly connect with the frontal. The stimulation of the cortex results in dilatation or contraction of the pupils in the course of an epileptic attack with which reaction the above is associated. We felt that not only the above and other autonomic symptoms in general, but also the following gastrointestinal reactions in particular consisting of chewing of the tongue, vomiting and the frequent defecatory reactions are also seen with many epileptics in the course of seizures. Later a description of the various forms of deviation of the eyes and head, with a presentation of the literature covering both experimental and clinical data pointing to various portions of the cerebral cortex aside from the frontal possessing areas representing motor functions involving head and eye movements as patterns in the course of an epileptic attack was offered.

Our presentation of cases will eventually fall along the lines of the data mentioned above. Without wishing to upset the literature on the clinical neurology of the cerebral functions given above, we will present material dealing with paralysis of the opposite half of the body as a consequence of lesions not only in the frontal lobe, but also in other areas of the cerebrum. In other words, if a patient suffers from a brain tumor in the occipital region and a paralysis of the contralateral side of the body ensues, this paralysis is not due as previously thought, indirectly, to pressure upon the precentral region, but to direct impingement upon motor areas further back in the parietal and even the occipital region itself. We feel that this multicortical representation for motion might include gastrointestinal and cranial nerve action. What has been said above of the occipital may also be said of the temporal and other portions of the cerebrum. Cranial nerve function and gastrointestinal neurogenic control by the cortex must, therefore, be firmly established over a wide area and this we intend to do subsequently by elaboration upon the cingulate system so that the mammillothalamic will not stand as an isolated entity. Thus we will interconnect the areas enumerated.

Without an attempt of presenting our rather ambitious designs for a revision of the accepted localization of functions in the motor area so that the functions of the same could be stretched to other portions of the cerebral cortex, we are induced to further fortify our position by presenting evidence that such revision is not altogether impossible. In the first place we have presented a great number of references to the literature to show that motor function, tonus, eye movements, etc. are localized in parts other than the accepted precentral region. We have also repeatedly reminded the reader of the existence of an intercortical system which we have given in detail and which we have found to be connected with many sulci and gyri that come in contact with the frontal region and are most likely to be connected with vegetative functions as well as eye movements, head movements, and motor functions and general tonus, and particularly that the cingulate region situated near these areas especially the endings of the mammillothalamic region is the most important one. The literature on the mammillothalamic region showed that fibres from the anterior nucleus of the thalamus on reaching the frontal cerebral cortex have been directly connected with the cingulate region among others. Although other fibre systems besides the cingulate converge into that portion of the frontal lobe concerned with gastrointestinal movement control, we feel that the cingulate is the most important of this group. Neither the arcuate nor the longitudinal system of the cortex have been shown to be directly connected with the hypothalamic system while the cingulate has been proven to be through the mammillothalamic tract. As we do not care to give a detailed account of the literature of this cingulate we refer the reader to Rosett. We will quote directly on this cingulate region, taken from various portions of his book on the intercortical system, so that we are not obliged to add more data from his bibliography to our already overburdened literature of the cortex.

The following quotations are taken from pages 33 to 36 inclusive: "In view of the great age of the cingulate fissure, which had its origin in that early period

of evolution when a new forebrain began to be superimposed upon the archaic rhinencephalon, one might surmise that it would appear correspondingly early in the human fetus. This is not the case. Transitory furrows and indentations, it is true, foretell the site of the future fissure as early as the fourth month of fetal life. They disappear, however, in the fifth month, with the mesial surface of the cerebrum smooth; and it is not before the sixth month of fetal life that several grooves and impressions appear which, by their various coalescence, build up the particular type of the permanent furrow. The reason for such a hard development of ancient structure is suggested by the following fact: The rhinencephalon, or the limbic lobe of the lower mammalian is a relatively compact structure. The unobtrusive band of the corpus callosum perforates it transversely from side to side. With the rise in the mammalian scale, there takes place an unequal increase

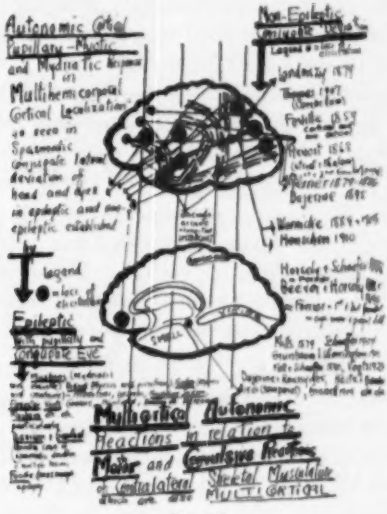


Fig. 30

in the size of the different parts of the cerebrum, such that the rhinencephalon lags behind. The corpus callosum, which increases in size in proportion to the increase of the new part, distends and distorts the original form of the rhinencephalon. This is evident in the course of the development of the human fetus. With every step in the progress of this distortion of the rhinencephalon and the diminution of its primary receptive apparatus, there has taken place a progressive increase in the number of pathways which connect it with the newer parts of the cerebrum. Great numbers of these pathways are deposited beneath the cortex of the cingulate fissure."

In offering Rosett's data we must impress the reader with the fact that of the intercortical systems we had the choice of the arcuate and longitudinal systems besides the cingulate. All three converge into the frontal region almost directly

into the area which Watts and Fulton designated as having neurogenic control of gastrointestinal function. From Watts and Fulton's viewpoint it might have been better to choose the arcuate system but from the data presented by the workers with the mammillary and preoptic systems it is evident that the cingulate system because of its accessibility to deeper structures and the mesial surface of the cerebrum is the tract of choice.

The following quotation is taken from Rosett (p. 35). It deals with the main facts about pathways of the cingulate fissure. "These pathways may be classed according to their different directions, under the heading of transverse, longitudinal and diagonal.

"1. Pathways which extend more or less squarely around the fissure between the outer and inner border may be seen on the map to cover it throughout its extent, with the exception of its rostral termination. In the situation of some of its tributary sulci, the transverse pathway exends beween the particular offshoot and the opposite order of the parent fissure.

"2. Longitudinal pathways extend either between the outer or the inner border of the fissure a.) the opposite border; b.) the posterosuperior tributary sulcus; c.) the posterior extremity of the subparietal portion of this cingulate fissure; d.) the anterosuperior tributary sulcus.

"3. Diagonal pathways: between the superior and the inferior borders of the subparietal termination of the fissure; between some of the offshoots and the opposite border of the parent fissure, between the opposite borders of some of the offshoots; between an inferior offshoot placed on the cingulum convolution (outlined not mapped on the picture) and two superior offshoots.

"*The pathways of a part of a cingulate fissure a two-piece type*:

"The variation of the fissure in which the posterior end of the pars anterior passes for some distance above and back of the anterior and of the pars intermedia was examined for the underlying intercortical pathways. The intervening convolution was shaved off after the piece was dissected out in the manner described, so that the fibre side of the fissure could be spread out on a glass slide. It was found that although many of the shorter fibre bundles were interrupted in the intervening convolution, at least two sets of longer bundles passed beneath this space in somewhat curved diagonals which crossed each other about the middle of their course from the point of intersection to the intercortical pathways, therefore the difference between a continuous and an uninterrupted type of fissure was in that case small."

With so many ramifications for the cingulate it becomes likely that not only the areas proposed by Watts and Fulton as carrying gastrointestinal motor function could be connected with the cingulate system but others as well. Temporal and parietal as well as postcentral areas subserving gastrointestinal function could be connected with the cingulate by remote control. Such areas including the motor cortex are nevertheless connected with the anterior nucleus of the thalamus via the intercortical and intrathalamic routes.

For further elaboration of the cingulate region see the other data on the subject by Rosett. The work of Rosett's intercortical system is relatively new and it will take a long time before the fibre tracts which he has established can be put into clinical use. Our presentation of the above quotation was however to impress the reader with the fact that one small fibre tract, by virtue of its location in a certain area, can become important because of the connections which it might establish. In this case the cingulate is connected with the mammillothalamic region and stretches posteriorly, entering the mesial surface of the cerebrum and in this way establishes other connections with distal portions of the hemisphere. This fibre tract will eventually become important because of its connections and

Fig. 31

Fig. 32

its cortical representation. In most of our writings we referred to the intercortical system almost as often as we did to Penfield's results with stimulation of projection areas. The importance of Rosett's intercortical system as we see it lies in the fact that some of the fibre paths which he elaborated upon consist of projection as well as association entities. The neuroanatomist is familiar with such divisions of fibre systems which are considered separately in works on the cerebral cortex in the Krause and Bailbarger lines.

In the above description of the extent of the cingulate fissure we see how a set of fibres can extend in all directions even though situated in the regions of the intercortical system, that is mainly below the upper layer of the cortex and surrounding the fissure beds. Rosett's monograph (1933) is characterized through-

out by a great deal of meticulousness with the subject of the intercortical system. In his attention to great detail Rosett shows a complete turnabout when he dealt with the scheme of pyramidal tract in relation to the autonomic. As we have repeatedly emphasized it is hard to believe this great cortical neuroanatomist should not have been cognizant of the fact that the autonomic within the cerebrum is much more extensive than he made it. His diagram makes us feel that he favored the neurogenic control of the autonomic by the final common pathway, that is the pyramidal tract rather than the nucleofascicular mammillothalamic or the corticosupraopticomammillary tract. In this respect he agreed with Watts and Fulton but particularly the former as well as with Spiegel and others. Another great authority, Archambault, who was a pupil of Charcot possibly seems to have favored the general representation of the autonomic system in the spinothalamic and related pathways constituting the pyramidal system. He was however much more elaborate anatomically, rather than encompassing in his collection of clinical material and more thorough than anyone who seems to have dealt with the subject in his localization of those structures which most probably carry cortical autonomic impulses. We shall present data from Archambault later when we discuss the problem of growth and trophic phenomena in general, in relation to muscular atrophy. Throughout our dealings with the neurogenic control of the viscera in relation to the gastrointestinal tract we were tempted to bring up the problem of growth. As is commonly known growth phenomena are invariably associated with disturbances in endocrine and metabolic function. Such disturbances are most often envisaged in the syndromes that accompany pituitary disease. It becomes evident on the basis of the above that the tuber cinereum and the paths situated in that locality as well as the various extensive fibres that are connected with the mammillothalamic tract may be responsible for the neurogenic control of trophic phenomena and growth in general. We know that to be the case in such conditions as acromegaly and pituitary cachexia, or what is known as Simmond's disease.

Although we were repeatedly tempted to digress into this fascinating subject of growth in its relation to localization within the cerebrum we nevertheless felt that it was important for us to implant in the mind of the reader the various tracts and other structures involved in the corticothalamomammillary nucleofascicular and supraopticomammillary tube, the function of which is the neurogenic control of the gastrointestinal tract. Although we could have referred a great deal more to the subject of mammillothalamic structures, we feel that enough has been accumulated in the foregoing pages to picture this as a definite pathway from the cerebral cortex of the frontal lobe downward to the anterior nucleus of the thalamus into the mammillary region so that the experimental evidence for its relationship to gastrointestinal and urinary function is being backed by a definite neuroanatomical entity.

Before considering the problem of neurogenic control of growth and atrophy we must at this point digress to the subject of the caudate nucleus. We have previously shown that Watts and Fulton claimed that motor functions of the gastro-

intestinal tract were controlled by the cerebral cortex through the extrapyramidal structures. If such is the case did they mean the caudate nucleus or the putamen and globus pallidus? If they are right with regard to this extrapyramidal route we feel that the caudate nucleus is the most likely structure for this function since its direct connection with the medial nucleus of the thalamus and hypothalamus gives us another mammillothalamic pathway. Although we have dealt with the subject of tonus in relation to the mammillothalamic tract in particular and the diencephalon in general, here and there we referred to its possible relation to the epileptic attack. We, for instance, gave a detailed account of Muskens' theory on the clonic stage in relation to the tonic stage during an epileptic attack. We also, in the recent pages, attempt to present material from the literature to show that in addition to the various parts of the brain involved in head, eye, neck and body movement during a Jacksonian attack, there was also a definite area in the motor and premotor region of the frontal lobe concerned with tonus. We were rather disappointed by, and repeatedly stressed the fact that Muskens, who probably has been the greatest authority on epilepsy during the first 25 years of this century, repeatedly insisted on relating his tonic phenomena and tonic transmitting structures to the corpus striatum. The problems of tonus and motion, whether related to the gastrointestinal musculature as presented by Watts and Fulton, or to the epileptic fit as proposed by Muskens, seem to be, according to these authorities, linked to the extrapyramidal pathways. Muskens felt that the corpus striatum controls hypertonicity of the extrapyramidal type and dwelt little on the pyramidal type of tonus. We ventured that conditions such as dystonia, chorea, and the rigidities of Parkinsonism were the conditions that were related to the globus pallidus and putamen rather than the hypertonicity during an epileptic attack. Of the abnormal involuntary movement syndromes that one classes with the basal ganglia, athetosis seems to be the exception from an anatomical point of view. Tilney and Riley seemed to be emphatic that athetosis is localized in the caudate nucleus. In our experience with many epileptics we find that athetosis seems to be the only symptom that is observed in epileptics of all forms of extrapyramidal syndromes. For this reason, but more because of the linking by some authorities of neurogenic control of gastrointestinal function with the extrapyramidal system, we will present the literature on the caudate nucleus after mentioning some of its functions.

We wish to impress the reader with the importance of the subject of athetosis in relation to the extrapyramidal system. This is the only striatal condition which occurs in epileptics although it is said to be caused by the lesion in the extrapyramidal system. As is well known it is a form of slow writhing motion and hence during this motion there is a variation in muscular tonus. Occasionally it is associated with chorea in a condition described as choreoathetosis. Both of these conditions are characterized by absence of terminal rigidity which is seen in so many conditions both of the extrapyramidal and pyramidal type. Because athetosis has been localized in the caudate nucleus by Tilney and Riley, we will later give a detailed presentation of the literature on the anatomy of this structure. Since tonus

is so important in the mammillothalamic system and in almost every condition associated with epilepsy it might be well to emphasize some of the points connected with tonus and body rigidity. In lesions of the spinal cord we find that abolition of tonus is associated with the flaccid paralysis of anterior poliomyelitis. Tonus seems to enter into spinal cord disease when the reflex is not completely abolished. We see altered tonus in extrapyramidal disease and always in involvement of the pyramidal system. Transsection of the cord is characterized by marked hypotonicity with paralysis below the level. Muskens very well discussed the problem of tonus and myoclonus in relation to epilepsy and referred to Cobb whose work indicated that the problem is dependent on a neural bioelectric phenomenon, consisting of partial interruption of the reflex arc without abolition of the same so that the possibility of conduction of potentials is still maintained. It is not unlikely that the bioelectric potential remaining is carried along various systems of fibres and cells within the central nervous system, and eventually manifests itself into types of rigidity. The extrapyramidal type of rigidity to which Muskens wished to connect the myoclonic phenomena consists of rigidity seen in Parkinsonism, dystonia and other conditions observed in encephalitis of the extrapyramidal type. The other type of rigidity is seen in pyramidal disease. It is present in multiple sclerosis, and in the rigidity of neurosomatic deterioration of epileptics, as well as in the rigidity of hemiplegia. When this type of rigidity becomes periodically spasmodic, we have an epileptic attack.

When the manifestations of tonus are carried in such structures that they become writhing, it may occur as one of the pyramidal or extrapyramidal diseases. If it occurs as the extrapyramidal conditions found in Huntington chorea then epilepsy does not supervene. Neither does it supervene when the familiar forms of extrapyramidal disease such as dystonia and paralysis agitans pictures are present. Epilepsy is however present in individuals suffering from athetosis. In the former conditions it seems that the potential which accumulates and would be used in the course of an epileptic attack (Muskens discharge method), has spent itself so that the attacks of toni clonus do not occur in the form of a regular seizure but as myoclonic shocks. That Muskens must be correct in the foregoing is also shown by the fact that in multiple sclerotics we rarely see epilepsy. In athetosis, however, epileptic attacks do occur. For the above reason it might be well to digress upon the subject of the caudate nucleus, which is one of the structures forming a basal ganglion which constitutes part of the telencephalon. Although we have repeatedly opposed the extrapyramidal system as having a neurogenic influence on gastrointestinal function we have at times been doubtful about our position. The anatomy of the caudate nucleus causes this doubt. This structure receives fibres from the hypothalamus and mesial portion of the thalamus. Such fibres pass through the dorsal thalamus on their way to the caudate nucleus. It is therefore not difficult to suspect that there might be a connection between the caudate and the mammillothalamic tract. This caudate structure is alleged to be

the organ, a lesion in which it is said to result in the extrapyramidal phenomenon of athetosis.

In cases with lesions causing the absence of cortical fibres the lenticular fasciculus or H2 of Forel was present (Probst, Von Monakow). Edinger and Fischer (1913), Wilson (1914), and Riese (1924b), claim that even though the greater part of the caudate and putamen was lacking in one side of the brain, this bundle (lenticular fasciculus) was equally well developed on both sides. Tarasewitch (1902) found the ansa lenticularis intact in a patient having a large injury to the putamen. Watts and Fulton were not specific enough when they claimed that gastrointestinal motor function was controlled by the extrapyramidal pathways. From their writings it is not evident whether they had in mind the corticorubral, corticonigral, the corticosubthalamonigral or the corticostriate fibre

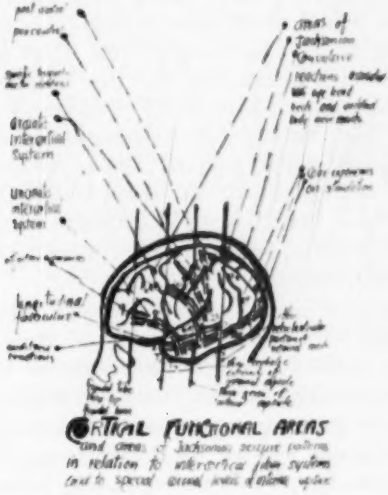


Fig. 33

systems. If they had in mind the third structure mentioned they would probably favor a mammillothalamic pathway by insinuation.

Since the caudate nucleus has been related in a number of syndromes to the lenticular nucleus and therefore to the ansa lenticularis as shown above, a number of experimentalists have attempted to establish a relationship between the caudate and the hypothalamus directly or via the lenticular nucleus. A number of observers have contributed to the knowledge of the fasciculus subthalamicus as a fibre tract originating in the nucleus subthalamicus. Among such authorities may be mentioned Nahaim, Von Monakow, Probst, Grünstein, Cecile and Oskar Vogt, Jakob, Riese, Foix and Nicolesco, Rioch and finally Kodama. The first two mentioned observers regarded this fasciculus as carrying putamenosubthalamic fibres. Kodama appears to have found such fibres. Probst, Grünstein and Riese were

unable to find degenerated fibres to the nucleus subthalamicus after an injury to the head of the caudate nucleus. Wilson (1914) regarded the fasciculus subthalamicus as arising exclusively from the globus pallidus. In addition to internuclear fibres connecting the subthalamic nuclei of both sides, certain observers have carried crossed fibres to the contralateral nucleus subthalamicus through the supramammillary commissure. Since many authorities feel that there might be a direct or indirect relation between the caudate lenticular and nucleus an indirect connection to the mammillary body is also seen from the above.

Edinger and Fischer (1913) state that they could find no degenerated fibres in the nucleus subthalamicus although the caudate and putamen were almost completely degenerated on one side. The functions of the caudate nucleus in relation to those of some of the other telencephalic basal ganglia is given by Kappers, Huber and Crosby. Our main reason for selecting the caudate nucleus and concentrating upon it instead of upon the lenticular nucleus is because the head of this nucleus and its other parts contain cells which extend into the medial nucleus of the thalamus. It is in this way that this structure is most proximally connected with the olfactovisceral part of the thalamus. Between such types of anatomical relationship and that of other thalamostriatal and striothalamic associations we can understand why Watts and Fulton favored the extrapyramidal system in their cortical neurogenic gastrointestinal scheme. It will be shown later by Archambault and Fromm that athetosis is associated with atrophy of the same extremity. The caudate nucleus seems therefore more related to the thalamus than to the lenticulostriate system.

The globus pallidus to which the caudate is related is believed to be a center for automatic associated movements. According to Hunt, Tilney and Riley, atrophy of the globus pallidus produces the syndrome of juvenile paralysis agitans. Wilson, Tilney and McKenzie and Tilney and Riley, claimed that Wilson's disease is a progressive lenticular degeneration in which the putamen particularly, but the globus pallidus also is affected. In the syndrome of double athetosis in which the caudate nucleus is particularly degenerated although the putamen is also affected, Tilney and Riley maintain that choreoathetotic movements are interpreted as an overabundance of automatic associated movements. Now that it has been established that the caudate nucleus connects with many structures including the medial nucleus of the thalamus and the subthalamus the question arises about another possible mammillothalamic pathway through the caudate nucleus which might carry gastrointestinal functions. This would definitely agree with the experiments of Watts and Fulton who insisted on an extrapyramidal path for such functions and add another corticothalamic route.

It becomes evident that the telencephalic basal ganglia are important structures which have for their function certain types of abnormal involuntary movements. The movements characterize the various syndromes of paralysis agitans, chorea, etc. Although Muskens was very much interested in these movements and particularly in the anatomy of this region, there is no direct evidence to show that the caudate is related to the hypothalamus as the len-

ticular nucleus is through the medium of the ansa lenticularis. That the caudate nucleus has its important connections, however, will be seen later. The balance of evidence at present favors the cortical regulation of striate function. Connections between the caudate nucleus and the cerebral cortex through the internal capsule have been reported by Cajal, Dejerine and Economo. Interrelation between the cerebral cortex and the caudate nucleus by way of projection fibres has been reported by Anton and Zingerle, Sachs, Von Monakow, Kodama, Coenen and others. The problem of the caudate putamen relationship to the subthalamic fasciculus is connected with the names of Bianchi and d'Abundo, Economo, Kodama, etc. as reported by Kappers, Huber and Crosby. Marisesco (1895) traced degenerated fibres from the caudate nucleus to the frontal lobe through the anterior limb of the internal capsule. Of all the anatomical contributions relating to neurogenic control of gastrointestinal function by the cerebral cortex this seems to be one of the most important. If the frontal lobe is connected with the caudate nucleus then Watts and Fulton were right when they maintained that gastrointestinal function is carried by extrapyramidal fibres. The caudate nucleus, being part of the extrapyramidal system and connected with the frontal lobe, connects below not only with the medial nucleus of the thalamus but also with the subthalamus and in this way probably reaches the mammillary bodies. Here we have of course another mammillothalamic system. Bianchi and d'Abundo, found that a lesion in the cerebral cortex resulted in the production of neuroglia in the caudate nucleus and putamen while the cells of the globus pallidus were proliferated and reduced in size. Additional data on the caudate nucleus may be found in Kappers, Huber and Crosby.

Throughout this work on the gastrointestinal system we must confess that we felt obligated to the gastroenterologist not so much because we lacked clinical material in the literature, or that we felt that the anatomy of the neurogenic control of the gastrointestinal tract was deficient; it was merely a matter of arranging the massive amount of material in the literature in such a coherent manner as to be of use to him. In the review of the material by Lennox and Cobb we were conscious of this deficiency. In our review of the data on the hypothalamus and mammillary regions we dealt largely with experimental material on the gastrointestinal tract and water metabolism. We also felt that if the material were properly divided into that which dealt with the motor function of the gastrointestinal tract and that which dealt with its secretory function, there was nevertheless some indecisiveness in regard to the destinaton of the fibres from the hypothalamic and mammillary region upward toward the cortex. Finally when we dealt with data by Watts and Fulton on intussusception as well as control of urinary function, we became more and more confused by neuroanatomical deficiencies. Watts and Fulton were blameless in not massing more literature on the anatomical basis of neurogenic control than they did. The literature has been too conflicting particularly in regard to cortical representation of gastrointestinal function. However, authorities such as Cajal and Dejerine shouldn't be neglected.

In order to give the gastroenterologist a clear picture of neurogenic control of the gastrointestinal system we must adhere largely to one tract, mainly the mammillothalamic tract. However, we do not want the reader to lose sight of the complexity of the autonomic structures throughout the cerebrum. For this reason we gave him a bird's-eye view of the probable shape of the fibre system and nuclear structures controlling gastrointestinal function. This we did in giving a description of a basket, the body of which forms a polyhedron. We showed that the mammillothalamic tract runs through this polyhedron and upward toward the cerebral cortex. As we went along we digressed here and there to show the reader that although we presented the neurogenic control of the gastrointestinal system along the mammillothalamic tract as a very simple structure, that its hook-ups below were really not so simple. Above the mesencephalon these structures within the diencephalic regions are characterized by a great many fibres, nuclei, commissures, decussations, radiations, peduncles, etc. all of which are connected with the mammillothalamic tract and with the anatomical polyhedron controlling vegetative functions at the base of the cerebrum. This shows the reader that the smallest lesion at the base of the cerebrum is likely to involve the autonomic nervous system above the diencephalon and therefore directly or indirectly effect motor and secretory control of the gastrointestinal system. We proved the above by giving a detailed description of the mammillary peduncle in the form of a citation from Ingram.

After acquainting the reader with the neuroanatomical problem dealing with the neurogenic control of the gastrointestinal tract by the cerebrum, we started by presenting the literature on the mammillary region and proposed to follow with the literature on the mammillothalamic tract by discussing the anterior nucleus of the thalamus following our discussion of the mammillary region. We felt that although Sheehan presented the problem of anatomy of the mammillary region and its intestinal tract rather clearly, his data was insufficient for our purpose. The division by him and the large mass of illustrious workers on this subject of the mammillary region dealt largely with the division of the hypothalamic region into preoptic, tuberal and mammillary groups. We had to supplement it with the record of cellular structure and fibre connections. We did this at length in previous pages as the reader knows, then we proceeded to the anterior nucleus of the thalamus and gave data on the cellular structure but concentrated greatly on its fibre connections. The fibre connections were very important as has previously been explained. For the experimental work of Watts and Fulton and the clinical material of the literature it was necessary that we show how fibres extend from the anterior nucleus of the thalamus upward into the cerebral cortex and into the areas which Watts and Fulton believed were responsible for the control of gastrointestinal motor function. Throughout our presentation of data along the above planned line of attack of the anatomical problem we were constantly aware of the fact that the lateral division of the dorsal thalamus is also concerned with vegetative functions. We purposely abstained from referring to this important structure because we felt that it

belonged to that group of workers such as Spiegel and others who maintain that neurogenic control of gastrointestinal function in the cortex is not confined to the frontal lobe but is mediated by the spinothalamic pathways whose neurones pass into the lateral nucleus of the thalamus. Evidently this is not the view held by Watts and Fulton, Keller and D'Amour or even Beattie and Sheehan. We also felt urinary functions which were related to water metabolism, although barely definite as far as anatomical localization within the hypothalamus, was very indefinite as far as cortical representation. Watts and Frazier showed this to be true. (Compare with data in Fig. 30).

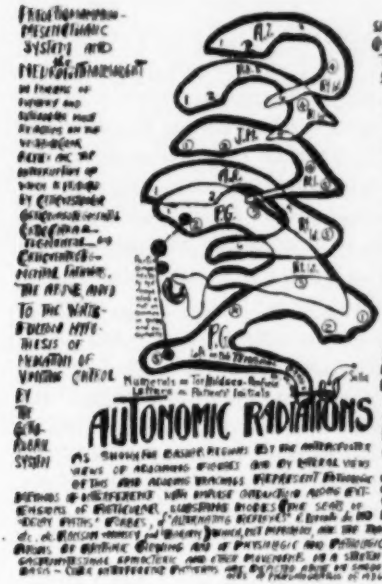


Fig. 34

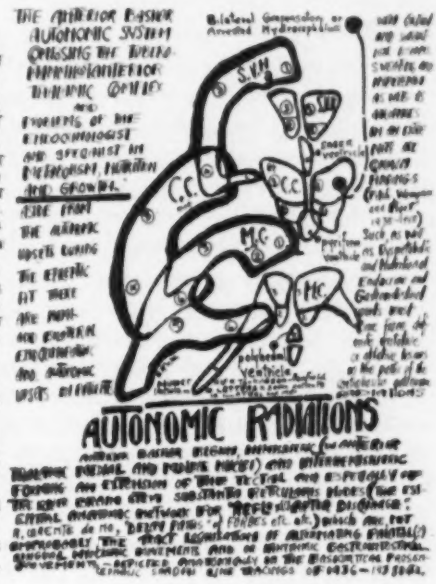


Fig. 35

After giving the fibre connections of the mammillothalamic tract as extending from the anterior nucleus of the thalamus into the cerebral cortex we felt that elaboration upon the cortical structures was very much in order. This elaboration was called for because of the indecision of Watts and Fulton and Frazier. These last mentioned authorities were so indecisive with regard to the exact localization of cortical functions controlling the gastrointestinal tract in other areas which they described that they never committed themselves with regard to whether they favored the mammillothalamic pathway or the pyramidal pathway. Because of the above we felt that it was necessary for us to impress the reader with the fact that although most of our anatomical and physiological material showed that the frontal lobe was responsible for the neurogenic control within the cerebrum, of

gastrointestinal and urinary functions, that this lobe was not the only one which contained centers subserving the same (Compare with areas in Fig. 30).

All are familiar with the fact that the neurogenic control of the gastrointestinal system depends upon sympathetic and parasympathetic representation. It is therefore logical to conclude that if other areas within the cerebral cortex show evidences of such activity that these might be related directly or indirectly to the cerebral neurogenic control of gastrointestinal function. While we are on the subject of the sympathetic within the cortex we might mention the cingulate gyrus in this connection. An excellent treatment of the subject is presented by Ward. He presents a large bibliography and summarizes the functions of this area in an excellent way. He states that electrical stimulation causes respiratory, cardiovascular and pupillary responses as well as piloerection. It also causes profound suppression of all motor activity (excluding heart and other smooth muscle?). Stimulation abolishes deep reflex and electrical activity of cerebral cortex. It may evoke complex bilateral tonic movement (intestinal?). Ablation causes loss of fear of man, social indifference to fellow monkey, loss of mimetic activity, grooming and signs of affection. Some of these functions especially that of feeling tone, etc. have been localized in other parts of the cerebrum in addition to the cingulate.

In our search for other areas within the cerebral cortex that might be centers of autonomic functions, sympathetic and parasympathetic, we did not wish to minimize the value of the frontal lobe, in fact, we repeatedly referred to the literature dealing with the frontal lobe and particularly with the region of the cingulate, sigmoid and other locations such as area 4 and area 6a, b to show the variety of frontal lobe functions which are aligned with the neurogenic control of gastrointestinal and urinary function. In referring to the cortical region in which the mammillothalamic tract begins, we showed that throughout the scale of the development of the cerebrum from the lowest forms upward, there have been evidences that the gastrointestinal tract was represented in the region stressed by Watts and Fulton, and by Watts and Frazier. In the lower forms and particularly in the vertebrates, and in the mammalian kingdom, the centers for rhythmic chewing and swallowing and the centers for emotional expressions of joy and anger, etc. were localized in the frontal region near the location of the beginning of authorities, feeling tone was related to this area where the mammillothalamic tract begins within the cerebral cortex. On frequent occasions we previously used the term feeling tone without proper explanation so that those who are untrained in psychology might have mistaken it for actual tonus. To feeling tone there is much more feeling than tone since it is more subjective than objective. In other words it is the afferent end result of any sensation or even mental experience that results in pleasantness or unpleasantness. Those who claimed that this phenomenon which is intimately related to the mimicosomatic reactions is localized in the frontal lobe (Tilney and Riley) have made a tremendous contribution. It means that affective expressive thalamic facial reactions are localizable in the cortex. We furthermore showed that although there is very little direct evidence with

regard to the control by the frontal cerebral cortex of secretory functions of the gastrointestinal tract that this is probably the seat of such control. (Compare areas in Fig. 30 with those of Fig. 33).

Since the frontal lobe is definitely credited with the functions presented above and in addition with certain areas which have for their control, disturbances in motion and tonus upon the production of a cortical lesion, it was felt that the problem of tonus should be considered in detail. This problem of tonus concerns the neurogenic control of the gastrointestinal and urinary functions as is well known. Although this phenomenon of tonus is considered by most authorities to have a localization in the region of the hypothalamus, or in the diencephalon, and therefore is particularly connected with the lower division of the cerebral representation of the mammillothalamic tract, we nevertheless felt that tonus whether of the smooth or musculoskeletal type should also be a function of the upper portion of the mammillothalamic tract, that is the thalamocortical division. Watts and Fulton stressed this point of the interrelationship of tonus in the musculoskeletal system and in the gastrointestinal system in their very interesting work on the sigmoid gyrus in relation to gastrointestinal function. For the above reason we dwelt in detail on the tonus of the pyramidal and extrapyramidal type and its localization in the cerebral cortex. Those individuals who localized feeling tone and mimicosomatic facial expression in the frontal region also confront this problem of pyramidal in relation to extrapyramidal. Does a lesion in the frontal lobe, which destroys the ability to emote essentially affect the corticothalamic pathway, and produce a blankness of expression because the facility for expression which is the function of that bundle is absent? If that is the case then the extrapyramidal system does not enter into effect. If, however, the blank expression is an extrapyramidal phenomenon then it means that the corticostriatal dominance ensued after the corticothalamic effect was obliterated. It so happens that this tonus as a phenomenon associated with convulsions of the Jacksonian type was also associated with pupillary dilations and contractions hence with parts of the brain that are related to the neurogenic control of gastrointestinal and urinary function (See Fig. 33).

Before proceeding to the problem of trophic phenomena relating to the mammillothalamic tract and particularly in relation to muscular wasting, or hypogenesis as seen in cases of diseases that are associated with the pyramidal tract, we will have to digress for some explanation. We have invariably been tempted to bring up the subject of growth in its relation both to the hypothalamic and therefore to the mammillothalamic tract as well as to other portions of the brain. Although there is a tremendous amount of material dealing with both growth and the mammillothalamic system in relation to the endocrine there is also a great deal in the literature on trophic phenomena that is not directly concerned with hypothalamic, tuberal, or mammillary region lesions. All along the data which we presented above, particularly the clinical material, we were repeatedly tempted to remark on ulceration of the intestinal tract as possibly being a trophic phenomenon related to muscular atrophy. Atrophy, whether of skin mucous mem-

brane, cartilage bone or muscle tissues of all varieties, is a trophic phenomenon. Everyone is familiar with pressure atrophy of bedsores. Every neurologist is familiar with the trophic skin changes seen in neurological conditions from syringomyelia to anesthetic leprosy. Trophic changes whether of peripheral or central origin, if neurogenic, are invariably due to autonomic disturbances. If some duodenal ulcer lesions are accompanied by atrophic and trophic neurogenic processes then the reader should realize, without our stressing it further, that there is a relationship between the analysis by Archambault and Fromm and our gastroenterologic problems. This possibility should be connected with the experiments of every investigator from Schiff to Beattie down to the present. We however did not feel right injecting remarks that would distract the mind of the reader from the subject matter. We mainly wanted to indicate to the reader that the gastrointestinal tract has motor and secretory function on a neurogenic functional basis. We shall dwell in detail, when we introduce the subject of muscular atrophy, upon the ideas that came to our minds with regard to trophic phenomena and growth in their relation to the mammillothalamic and other tracts. After we introduce the subject of growth we shall spend an extensive amount of space on the paper by Archambault and Fromm in the hope that the reader will see that a subject such as that involving the neurogenic control of trophic phenomena in both smooth muscle and skeletal muscle is a very important one. Before we embark upon this very illuminating paper which we intend to quote and refer to extensively, there are a certain number of points with which we must impress the reader. These points have to be mentioned at this time because one of them involves the paper which deals with muscular atrophy.

We feel at this time guilty of three rather important transgressions. The first one is the fact that in such an extensive neuroanatomical discussion of the literature we include clinical material instead of adhering to laboratory and anatomical material exclusively. Anatomical material so long as it deals with autopsy findings seems to be in line with the subject of anatomy of the neurogenic control of the gastrointestinal tract. We confess that here and there we did include clinical material on the subject of anatomical discussion instead of adhering to laboratory degeneration experiments and pathological data. In most of our discussions on the neuroanatomy of the mammillothalamic tract, we only digressed once to present material which is essentially clinical. This material was in the paper by Davison and Goodhart on the spasmodic conjugate lateral deviation of the eyes. Although this paper was largely a clinical one there was some material in it which had pathological and neuroanatomical illustrative data. Over 80 per cent of our references on the subject of the neuroanatomy of the corticodiencephalic tract consisted of references that were not of a clinical nature. These references adhered to the subject of neuroanatomy and almost to that alone.

The second transgression of which we feel guilty was that we seemed to favor the mammillothalamic tract as being the only tract responsible for the neurogenic control of gastrointestinal and urinary function. We admit that we did this intentionally. We intend however to remedy this overemphasis on an anatom-

ical pathway of such an important subject as the autonomic nervous control but we propose to remedy this by spending a great deal of the remaining space left to us on the subject of neuroanatomy, confining ourselves to the final common pathway. We will therefore devote ourselves to the paper by Archambault and Fromm in great detail only because we have failed to impress the reader with the fact that the final common pathway contains both pyramidal and extrapyramidal fibres both of which, but most probably the former, have trophic function and are intimately connected with the problem of growth. In our future dealings with the problem of muscular atrophy the reader will be convinced that there are a great many neurological diseases involving the pyramidal pathways and associated

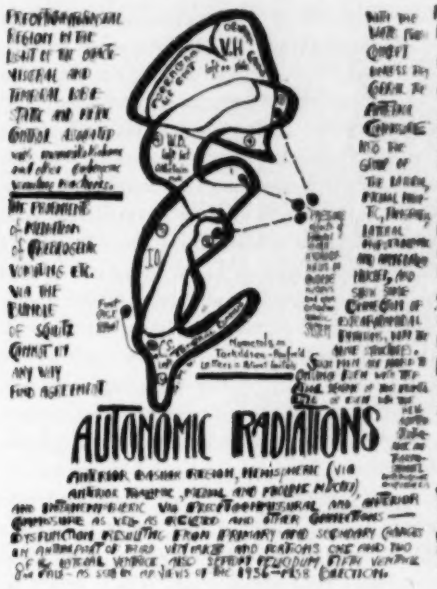


Fig. 36

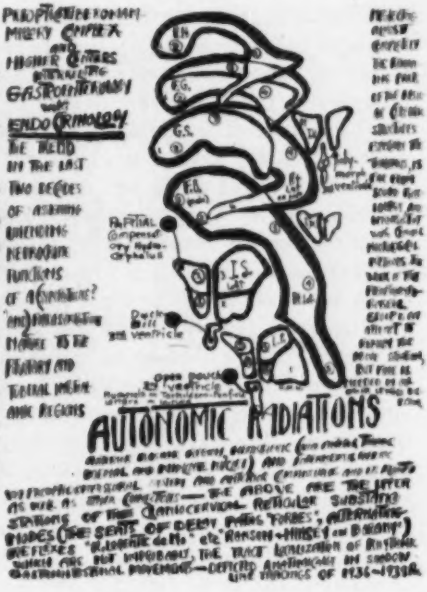


Fig. 37

with paralysis as well as muscular atrophy that show that the final common pathway is a very important one as far as trophic phenomena is concerned with muscular and osseous structure. We will in fact begin our reference to Archambault and Fromm by citations from the literature and emphasize that these authors completely omitted the extrapyramidal system.

The third criticism which we feel is the most important one is the fact that in our paper up to now, we have confined too much of our attention to experimental material which might include lower forms not approximating the human as far as the anatomical structures of the mammillothalamic tract is concerned. It may, in fact, be said that most of the material which we have presented heretofore, with the exception of the paper by Lennox and Cobb, dealt with experimental work done on animals. We find that the paper by Sheehan and that by Watts

and Fulton as well as that by Watts and Frazier all contained a great deal of material, which was largely experimental, on various forms of mammals. These papers from which we drew much of our references contain however a great deal of clinical and pathological data such as inflammatory lesions and tumors in the human brain. These lesions were definitely related to the mammillothalamic tract and various portions of the same which we have outlined in our scheme of the corticodiencephalic system. This evidence of human material found in the papers of the authors just mentioned also characterized our references to the literature which we obtained from Kappers, Huber and Crosby and the data on water metabolism.

We admit, however, that an appreciable amount of our neuroanatomical data consisted of anatomical facts which had to be obtained by degeneration experiments on various forms. The animals included were mostly mammals. In most instances we tried to present data on apes so that the anatomical structures could approach those of the human as closely as possible. Although the data which we presented on the lower portions of the mammillothalamic tract contain a rather extensive reference to the literature in which the human material consists of various lesions in the hypothalamus and surface of the cerebrum, we nevertheless confessed that on our presentation of the cortical data as seen in the papers by Watts and Fulton, and Watts and Frazier there was not quite as much material as that which we presented in the paper dealing with the hypothalamic and related areas. The reader might say that we should have confined ourselves almost exclusively to material of an experimental nature that dealt with the higher forms such as the monkey and related animals. Our answer to that point is the fact that we were anxious to follow a certain scheme of corticodiencephalic neurogenic gastrointestinal control. We were anxious to illustrate the extensiveness of the diencephalic autonomic centers. For this reason we presented a bird's-eye view of the anatomy of the subject under consideration in the form of a basket on a tripod. In order to give the various structures of the diencephalon and follow a scheme such as prethalamic, epithalamic, retrothalamic, mesothalamic, subthalamic, etc. a great deal of material has to be included as experimental data on animals which tends to support the assignment of specific functions to the various structures in such a scheme. There was therefore too much material for us to confine ourselves to experiments which dealt with the higher forms of life. In our discussion of the mammillothalamic system we however attempted to present data which not only dealt with human pathologic material as much as possible, but experimental data which dealt largely with experiments upon the higher forms such as monkeys and other species in the upper mammalian scale.

We could have been much guiltier if we indulged in the monumental references to experiments on dogs as carried out by I. P. Pavlov. For thirty years this great physiologist devoted his experimental work to dogs. This experimental work dealt largely with the relationship of cortical lesions to various functions of the gastrointestinal tract. Our bibliographic references could have been quite bulkier if we included all the material of the literature which had a bearing on the neuro-

genic control by the cerebral cortex of gastrointestinal function. We feel in fact that Watts and Fulton made a gross error in not giving a detailed analysis of the various types of cortical lesions produced by Pavlov in his experiments on dogs. Although Pavlov's experimental work did not deal with the neurogenic control of actual motor phenomena within the gastrointestinal tract, his data more than covered the cortical control of secretory functions of the stomach, intestines and salivary glands. Of particular interest is Pavlov's investigatory reflex. It forms the basis of volition and attention and seems to involve almost every sphere of sensory perception from touch, vibration, pain and kinesthesia, smell, taste, vision and hearing. There is only one place in the entire nervous system which could, in a reflex manner, fulfill all of these requirements and be at the same time associated

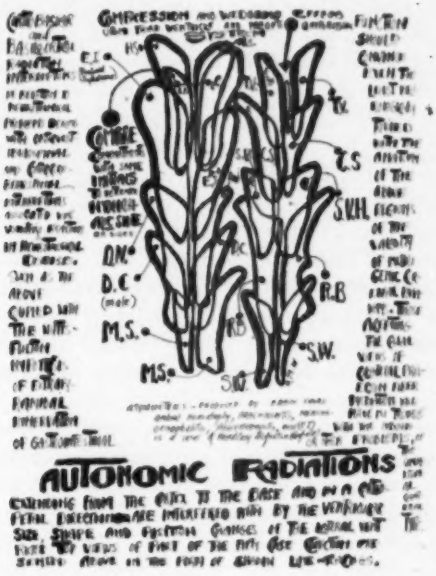


Fig. 38

with visceral operations. This place is the dorsal thalamus containing within it the anterior nucleus of the thalamus which is connected with the thalamomammillary and supraopticomammillary system. Although Watts and Fulton did not take advantage of the work of Pavlov, we feel that some day some neurologist will attempt to correlate lesions in the human, and relate them to gastrointestinal function.

Although Pavlov's work could have been included in our references to the anatomical literature in spite of the fact that he worked on forms within the mammalian kingdom which are below that of the human scale, his data would have served us only in part. It must be remembered that Pavlov in his work on the salivary glands in relation to certain functions connected with the cerebrum con-

fined himself largely to functions which are essentially cortical. He spent most of his time connecting the relationship between the perception of certain sensations such as sound, touch, pain, vibration, etc. to the amount of secretion of saliva and to the complete inhibition of the same. Pavlov, who was primarily a physiologist and of such renown that mastery of neuroanatomy by him should never be subject to question, evidently did not care to be explicit with regard to the exact conduction path followed by his salivary reflexes. One can see from his writings that he did not confine himself to olfactory experiments. He used every avenue of centripetal approach including kinesthetic sensations. Considering the above we feel that he probably thought that the spinothalamic and other pathways leading into the lateral nucleus of the thalamus carried stimuli which could affect salivation. Whether this is produced because of the connection of this nucleus with the medial or olfactovisceral nucleus Pavlov did not state. Although Pavlov took great pains to connect motor functions of the animals in their relation to salivary secretion and even showed that the emotional reactions of his dogs had a bearing upon the amount of saliva secreted, his material was largely cortical. In other words he never attempted to show how the lower portion of the mammillothalamic tract such as the anterior nucleus of the thalamus, the hypothalamic regions and particularly the mammillary regions influenced by lesions produced within them the activity of the salivary gland or that of the mucosa of the stomach and intestines. A review of the work of Pavlov will show here and there that extirpation experiments were performed in which parts of the entire cerebral lobe were removed and the functions of the animal were studied in relation to salivary secretion in the absence of that portion of the brain. In the matter of extirpation experiments, but in these only, it could be said that Pavlov experimented upon deeper structures.

Pavlov's interest in the cerebral cortex in relation to salivary and stomach secretion could be used in physiological experimental observations as a reference work. It is, however, very much neglected because of the psychological implication that Pavlov's experiments produced. The value of Pavlov's work on a gastroenterological basis has therefore been lost sight of. Although the Pavlov school dealt with visual, tactile, auditory and other stimuli these were invariably employed in relation to salivary secretion. Gastrointestinal glandular reactions were employed for study by this school of pioneers in relation to such centers as sleep and heat regulation. These workers did not stress the diencephalon for such gastrointestinal function, but the cerebral cortex. Secretory and trophic phenomena whether under cortical or diencephalic control have such far reaching repercussions in medicine as may be seen in duodenal and gastric ulcer studies that any and all data relevant to the neurogenic control such as will be seen in the work of Archambault and Fromm is almost indispensable. It is rather unfortunate that the gastroenterologist has been so handicapped by the lack of data on neurogenic control of the gastrointestinal tract that he has been steered away from material such as that presented by Pavlov. His use of dogs instead of monkeys is a serious objection. Note how the data in Figures 30 and 33 apply to Pavlov.

*(To be continued)*

# EDITORIAL

## ERRORS OF PRIORITY CREDIT IN MEDICINE

### Forgotten Names

*"But these are deeds which should not pass away, and names that must not wither"—Byron*

*"Live as to die tomorrow*
*Learn as to live forever"*
*Isidore of Seville Ca. 630 A.D.*

Garnett Cheney and others in California, where cabbage must grow abundantly, noted that in guinea pigs, cabbage appeared to prevent the formation of ulcers due to histamine. In eleven of 13 cases treated with cabbage juice, craters disappeared within nine days. Each patient drank a liter of fresh uncooked cabbage juice daily. Average healing time for seven cases of duodenal ulcer was 10.4 days, and for 6 cases of gastric ulcer 7.3 days. About 4½ pounds of cabbage yields one quart of juice. Celery juice, tomato juice, salt and pepper may be added to improve the taste. What the Stanford University group apparently did not know was that Marcius Percius Cato (234-149 B.C.) of Tusculum, and Rome, in his "De Re Rustica" recommended his favorite vegetable, cabbage, for the sick, for stomach and bowel trouble, and he even injected cabbage juice into sinuses and fistulas by means of a syringe composed of a bladder tied to a reed. He prescribed cabbage internally and externally, raw or cooked. Pliny and others also prescribed cabbage and other vegetable juices. Cato also applied mashed cabbage locally to ulcerated areas.

Aesclepiades of Bithynia practiced "psychosomatic medicine". He advocated change of diet, frequent baths, massage and exercise. He insisted upon kindness to his patients, and he was among the first to treat mentally diseased patients with gentleness. Aesclepiades ordered his mental patients to be moved into the sun, provided with music and songs and forbade them to be incarcerated in dark cells. Pliny and Galen, however, were his bitter critics.

It is of interest to quote Samuel Fenwick: "It will be readily conceded that the anemia that accompanies atrophy of the stomach is the result of the imperfect secretion of the gastric juice consequent upon it. Iron is rarely of service in the severe anemia associated with atrophy of the stomach."

Galen in his "De Affectorum locorum notitia, libri sex", Henricus Stephanus 1513, Paris—(Under "Hepatis Affectus") in discussing "Scirrus Hepatis" Liber V, Caput VI, (obverse) page 107, lines 23-28 states ". . . Since the liver is the chief instrument of the blood-making faculty, it follows that if this organ is weakened or diseased, that, also the function of this organ will be impaired or vitiated".

Alfonso Pirera (1877- ) of Naples, working in Castellino's Clinic bled dogs and rabbits to varying degrees of anemia. He then gave them liver orally, subcutaneously, intravenously and intraperitoneally. He used liver extract, and liver juice and found prompt and favorable erythropoietic response. Pirera also observed the stimulating effect of liver injections on the leucocytes, particularly the

granulocytes. Paul Carnot, A. Gilbert, E. Vidal of Paris, Maurice Perrin of Nancy observed the favorable effect of liver therapy and published their observations nearly 50 years ago.

## Psychosomatic Medicine

Socrates (469-399 B.C.) came back from army service to report to his Greek countrymen that in one respect the barbarian Thracians were in advance of Greek civilization. They knew that the body could not be cured without the mind. Socrates exclaimed "this is the reason why the cure of many diseases is unknown to the physicians of Hellas, because they are ignorant of the whole".

"Just as you ought not to attempt to cure eyes without head or head without body, so you should not treat body without soul".

Hippocrates (460-344 B.C.) said: "In order to cure the human body it is necessary to have knowledge of the whole of things". Shakespeare in King Lear (Act. II, Scene 4), says Lear—"We are not ourselves when nature, being oppressed, commands the mind, to suffer with the body" and in Macbeth (Act V, Scene III):

"*Macbeth*: Cure her of that.
Canst thou not minister to a mind diseas'd,
Pluck from the memory a rooted sorrow,
Raze out the written troubles of the brain,
And with some sweet oblivious antidote cleanse the stuff'd bosom of that perilous stuff,
Which weights upon the heart?
"*Doctor*: Therein the patient must minister to himself.
"*Macbeth*: Throw physic to the dogs, I'll none of it."

Paracelsus (1493-1541) said: "True medicine only arises from the creative knowledge of the last and deepest powers of the whole universe; only he who grasps the innermost nature of man, can cure him in earnest". One should give thought and consider Plato's "Eros", Aristotle's "Orexis", the work of Sigmund Freud, Spinoza, Sherrington, W. B. Cannon, A. Meyer, McKinney, Franz Alexander, Felix Deutch, and the publications of Jacob Kaufmann of New York, and Usher Parsons, of Providence, Rhode Island (1840), of John Actuarius the Greek (died 1283), and, of Erasistratus, (Ca. 310-250 B.C.) a grandson of Aristotle (384-322 B.C.) who visited the Schools of Metrodorus, Theophrastus and Chrysippus. Erasistratus, settled in Alexandria, but, before going there, he had occasion to treat Antiochus the son of King Selencus Nicator (or Selencus I, the founder of the Syrian kingdom of the Selenciadæ) during the period of about 290-280 B.C., nearly twenty-two hundred and fifty years ago. According to Plutarch, Selencus, past middle age with a grown-up son Antiochus of Apama (born in Persia) solicited Stratonice's hand in marriage. She was the daughter of Demetrius Poliorcetes and Phila, Antipater's daughter. She was not yet twenty years old when old King Selencus I married her. Antiochus fell madly in love with his young beautiful stepmother and "lived in the greatest anguish and affliction, fight-

ing with all his force against his passion, and realizing how abnormal his desire was, and how intolerable his suffering, contemplated suicide". He became seriously ill and his life was endangered, to his father's despair, since nobody was able to diagnose Antiochus' illness or to give him relief. Erasistratus understood that Antiochus was in love and by carefully studying his lack of reactions to other good-looking girls, but great changes noted when Stratonice appeared alone or with Selencus, he concluded that this was the cause of Antiochus' illness. Erasistratus so reported to Selencus, and so he proclaimed Antiochus king of all the Upper Provinces and Stratonice his queen, the two being married. Thus, Antiochus

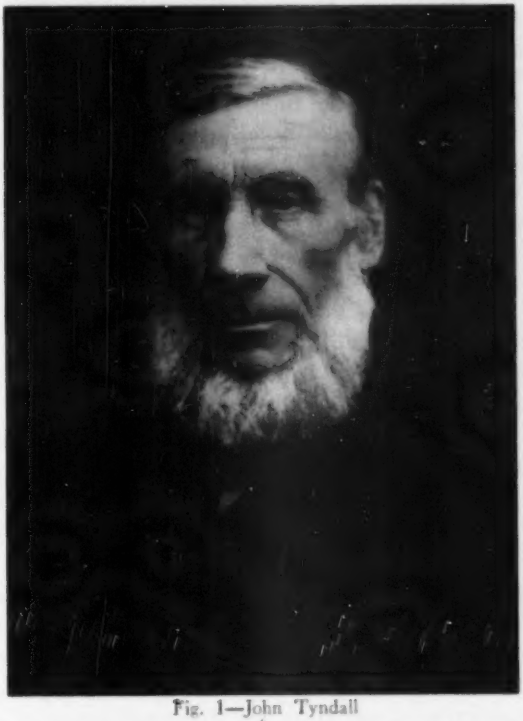

Fig. 1—John Tyndall

was cured, through Erasistratus' early knowledge of psychosomatic medicine (C. Alberto Segius).

Quoting Maudsley, "The sorrow that has no vent in tears may make other organs weep."

## Antibiotics

In his "Essays on the Floating Matter of the Air in Relation to Putrefaction and Infection", John Tyndall (1820-1893), working in his laboratory in 1874-1876, found that the "clay" of thick blanket of pencillium glaucum caused the bacteria to become quiescent and dormant and die producing the "slime of dormant

or dead bacteria". "I found no active life in this tube (with penicillium glaucum) while all the others swarmed with bacteria". On page 59-60 Tyndall says: "Penicillium glaucum . . . covered the entire surface with a thick tough layer, which must have seriously intercepted the oxygen necessary to the bacterial life". The bacteria lost their translatory power, fell to the bottom and left the liquid between them and the superficial layer clear. Six other tubes . . . became thickly covered with mould, under which the bacteria died or passed into a quiescent state, fell to the bottom, and left the liquid clear". These observations were made in 1874 and 1875. Tyndall, apparently communicated some of his findings, by an exhibit of his samples, to the Royal Society of London following January, 1876.

P. Vuillemin in 1889 coined the term "Antibiosis"—the active participant will be the "antibiote" (Antibiotics of today). Sir Alexander Fleming of London says: "The name of 'Penicillin' was given by me in 1929 to an antibacterial substance produced by a mould of the genus Penicillium. Penicillin belongs to a class of antibacterial substances produced by living organisms which as far back as 1889 were termed "Antibiotics". Sir Howard Florey of Oxford University and Sir Alexander Fleming of St. Mary's Hospital, University of London do not mention in their early publications the early reported studies of John Tyndall (1874-1876) with Moulds, and Penicillium Glaucum and its effect in killing or rendering bacteria "inactive and quiescent".

The name Penicillium applied to a genus of fungi first apears in Link's "Observationes" (1809) in which he described very briefly the genus and three species: Penicillium Glaucum, P. Candidum, and P. Expansum.

In 1824, he applied the name P. Glaucum to all green Penicillia including rot of apples and related fruits. In 1874, Brefeld published the life history of Penicillium glaucum. Pencillium notatum with which Florey and Fleming worked to give us the miracle antibiotic penicillin was first described by R. Westling in Upsala, in 1911 (forty years ago)—a green species of penicillium.

The Chinese, 2,500 years ago, were aware of the healing properties of moulded curd of soy bean when applied to carbuncles, boils, and like infections, and they used this mould treatment as the standard treatment for such conditions.

Pasteur and Joubert (1877) early recognized the clinical potentialities of micro-organisms as therapeutic agents as well as causal agents in disease.

## Angina Pectoris and Coronary Artery Disease

Did not Jean Baptiste de Senac (1693-1770) of Versailles, physician to Louis XV, chief physician to the Duke of Orleans in 1752, and who treated the Dauphin while he was physician to Louis XV, make observations on instances of congenital heart defects, and describe cases of coronary sclerosis (without painful seizures) and, marked thinning of the left ventricular wall (ventricular aneurysm) associated with coronary sclerosis? Did he not comment on the use of quinine in "rebellious palpitations" (auricular fibrillation?) under the caption "Operation of

stomachic remedies in palpitation, including the use of quinine in "rebellious palpitation". ("Traite de la Structure du Coeur", 1749)—200 years ago?

Did not Sir Thomas Lauder Brunton (1844-1916) of Edinburgh and London observe the prompt relief in angina pectoris on the use of amyl nitrite by inhalation (1867)? One recalls here, too, that W. Murrell reported the use of nitroglycerine as a remedy for angina pectoris (1879), while M. Hay introduced the use of sodium nitrite in the treatment of angina pectoris (1883). Of interest, too, is the Bradshaw Lecture on Some New Vasodilators, by J. B. Bradbury just fifty-five years ago.

Today we use quinidine with which Karel F. Wenckebach of Vienna restored normal rhythm in auricular fibrillation. In 1914 his associate W. von Frey, also found that quinidine was effective.

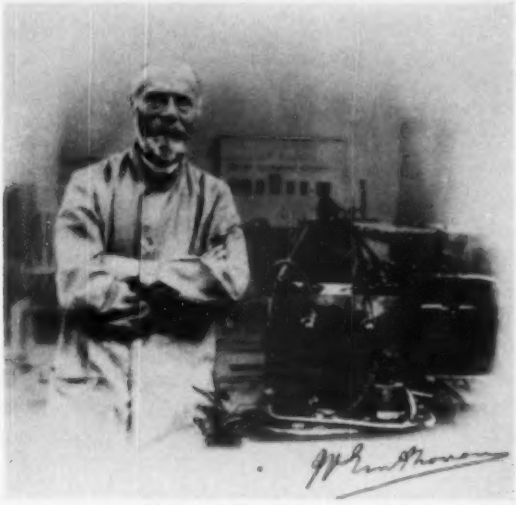

Fig. 2—Willem Einthoven

It was van Heijningen in 1849 who first isolated quinidine.

It was Sir John Burdon Sanderson (1828-1905) of England with F. J. M. Page, who first recorded the electrical activity of the heart by means of the capillary electrometer 70 years ago (1879-1880), followed by A. D. Waller (1856-1922) in 1888. Then came G. Fano, followed by Willem Einthoven's (1860-1927) (1903) "electrocardiograph"—a light and extremely delicate string galvanometer, and the most helpful contributions by Sir Thomas Lewis (1881-1945) and Sir James Mackenzie (1853-1925), the work of Carl Julius Rothberger and Winterberg, of Vienna, and Frank Wilson, Smith, Levine, H. B. Sprague, White, Pardee, J. B. Herrick, Katz and others in the United States, and John Parkinson and his Associates in London and Charles Laubry and his co-workers in France, and the late

Wenckebach (1864-1940), and Dressler, Scherf, and Roesler formerly of Vienna, and now in the United States.

But, lest we forget, Carlo Matteucci (1811-1868) of Pisa and Forli, Müller's pupil, in 1842-3, observed that electric currents were produced by cardiac contractions (in frogs and pigeons and other animals), as did also von Kölliker (1817-1905) and Müller in 1856. Also, that it was Alfred Einstein Cohn (1879- ) who was, perhaps, among the first to describe the effect of digitalis on the T-waves of the electrocardiogram. The discovery (1893) of the auriculoventricular bundle by William His, Jr., (1863-1934), deserves mention.

Leon-Louis Rostan (1790-1866) in his "Memoire sur les ruptures du Coeur", in 1820, stated that the scars found on many hearts indicate healed rupture (myocardial infarction), and he no doubt observed cases of coronary occlusion with myocardial infarction, and rupture.

Frederick Winsor (1829-1889), at the meeting of the Middlesex East District Medical Society held at his home in Winchester, Mass., March 24, 1880, after supper, read a paper on "Angina Pectoris with Rupture of the Heart". He states: "The attack in Albany, a fortnight before death, of severity before unknown, and accompanied by marked rigor, may have resulted from occlusion of the left coronary", . . . "that, from that time degenerative action was set up in that part of the ventricle no longer nourished by this artery, and that rupture did not occur till just before death". The necropsy was performed by Dr. W. F. Whitney, confirming the occluded left coronary and the sclerosed right coronary artery. The left coronary artery was found to be badly sclerosed, the inner surface of the walls of the artery was covered with yellowish-white patches, and, at one point, 3 cm. from its origin, one of these patches had so far protruded into the lumen of the vessel as to cause a coagulation of blood (coronary thrombosis) at that point, which coagulum had become adherent to the walls, thereby preventing the flow of blood through the vessel". This report by Winsor (1881) and the reports of other cases, by discussers at this meeting, and the reports by Adam Hammer (1878), Rostan (1820) and William Harvey in Littera Altera (Secunda) to Jean Riolan, of the case of the Nobleman Sir Robert D'Arcy, and a case report of spontaneous rupture of the left ventricle in the famous case of young Lord Montgomery followed (1660, posthumously).

It was Niels Stensen (1638-1686) who proved the muscular nature of the heart in his famous "De Musculis et glandulis observationum specimen, cum epistolis duabus anatomicus", Amstelodami, P. le Grand, 1664.

May I recall, that, Archangelus Piccolomini (1526-1586) in his "Anatomicæ Prælectiones Explicantes Mirificam Corporis humani fabricam, et quæ animæ vires-Romæ, B. Bonfadini, 1586—said: "I am of the opinion that the obliteration (or obstruction) of the little cord or vessel (or nerve) which goes to the heart, suddenly brought on, is the cause for which some persons go suddenly and unexpectedly out of this world". We must realize that many life-saving observations

were made by wise and experienced physicians and investigators in the decades and centuries before our era. It is tragic indeed, that many of us have ignored the writings of the great men of the past and, the literature of the world before our time, so that we missed the value of Piccholomini's observations, of John Tyndall's observation, of the work of the Frenchmen and Italians who recognized the value of liver therapy, long before we finally realized that the antibiotics of Fleming,

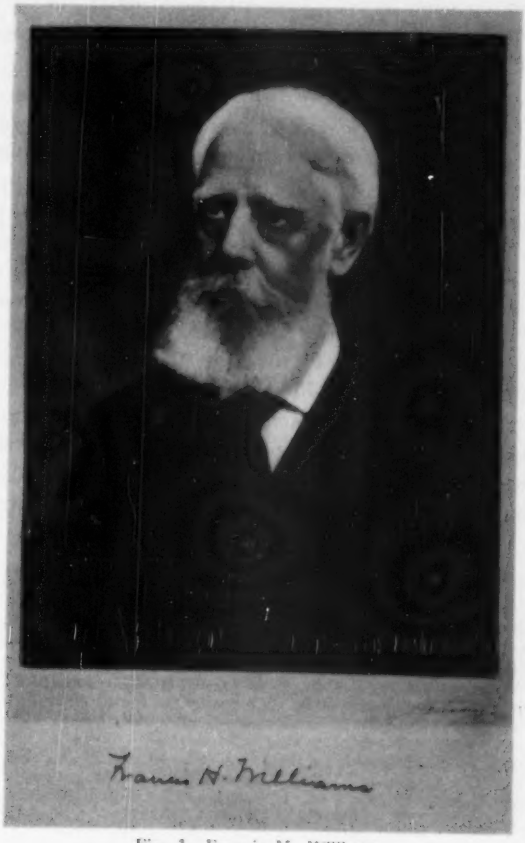


Fig. 3—Francis H. Williams

Florey, and Waksman, and others, and the liver therapy of Minot, Murphy, and Whipple, and the knowledge gained from studying the electrocardiographic tracings may lead to the saving of thousands of lives. Yes, with Tennyson, let us repeat "Most can raise the flowers now, For all have got the seed".

How many of us recall that Johannes S. C. Schweigger (1779-1857) of the University of Halle invented the string galvanometer? How many of us know that the electrophysicist Matteucci (1843) proved by experiments on frogs' and pigeons'

hearts that muscle action produced electric currents? That Albert von Kölliker (1817-1905) of Zurich and Würzburg and H. Müller established the occurrence of action currents resulting from cardiac contractions with each beat? How many of us know that Marcelli Malpighi (1628-1694) of Bologna in his two letters to Borelli (1661) described the vesicular nature of the lungs and was among the first, perhaps, to demonstrate the anatomic basis for the actual conception of the process of respiration (first letter)? That his second letter gave, perhaps, the first description of the capillaries and capillary circulation (1661), using Felice Fontana's (1646) microscope with a positive eyepiece.

We all know of Harvey's (1628) discovery of the circulation of the blood, of Matteo Realdo Colombo's (1516?-1559) description of the pulmonary circulation (but not conclusively), of Andrea Cesalpino's (1520?-1603) observations on the circulation of the blood, and the "discovery" of the pulmonary circulation by Michael Servetus (1509-1553) in Restitutio Christianismi. But, why have we overlooked or forgotten, the first discovery of the pulmonary circulation by an ancient Arabian physician of Cairo, Ibn-an-Nafis (ca. 1210-1288) (in his commentary on Avicenna's Anatomy)? Let us recall that a Veterinarian, H. Bouley (1831) observed intermittent claudication in a horse, and Goubaux (1846), and Charcot (1858) observed it in man giving us the term "intermittent claudication", while Erb called it "intermittent limp". Lobstein in 1833 gave us the term "arteriosclerosis" as in modern usage. Quoting Dr. Don José de Letamundi of Spain "El medico que no sabe mas que medicina, ni medicina sabe". (The doctor who only knows medicine, does not even know medicine), and Cicero: "History is the witness of the times, the torch of truth, the life of memory, the teacher of life, the messenger of antiquity".

HYMAN I. GOLDSTEIN.

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# ABSTRACTS

## Stomach

### PERFORATED GASTRIC ULCER ASSOCIATED WITH SUBCAPSULAR HEMORRHAGE OF SPLEEN. J. A. Shepherd. Brit. M. J. 1082-1083, (June 18), 1949.

This article deals with the report on a case of perforated gastric ulcer in a 69 year old woman. The ulcer perforated 11 hours before admission to the hospital. No vomiting developed subsequent to the perforation. This woman had a peptic ulcer syndrome for 40 years. Her abdomen on admission presented the usual findings of peritonitis.

At operation a large perforated ulcer on the anterior surface of pars cardiaca was found and repaired. The spleen was friable and surrounded by blood and clots. No thrombosis of the splenic pedicle vessels was observed. The laboratory report on the splenectomized specimen revealed no macroscopic evidence of infarction but a diffuse subcapsular hemorrhage was present and microscopic examination revealed a necrotic capsule with sclerosis of the splenic vessels.

The author points out the danger of adoption of conservative treatment for perforated peptic ulcers. He cites several other cases where similar subcapsular hemorrhage of the spleen developed in perforation of peptic ulcers.

A. X. Rossien

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## Intestines

### MULTIPLE POLYPOSIS OF THE COLON. G. H. Schantz. Gastroenterology. 13:430-439, (Nov.), 1949.

Multiple polyposis was noted in 1721 by Menzel, and Luschka in 1861 gave a convincing report of a case of multiple polyposis of the colon in a 30 year old woman. Virchow in 1863 gave an accurate pathologic description of "colitis polyposis cystica".

Harrison and Cripps in 1882, showed the familial relationship of this condition and reported 3 cases occurring in one family. Polyposis of the colon is often seen to run in families, and these polyps tend to become cancerous.

The disease can be diagnosed by rectal and sigmoidoscopic examination and barium double-contrast enemas.

All members of a "polyposis family" should receive thorough and repeated examinations. Almost all recorded cases of multiple polypi of the colon eventually become malignant (Lockhart-Mummery). The onset of this disease may be entirely insidious and be entirely lacking in symptoms! The treatment of the condition is strictly surgical and one must eliminate the entire mucus-bearing area of the colon.

The author reports typical familial cases from the St. Luke's Hospital, Bethlehem, Pennsylvania, seen and treated between 1928 and 1944.

Hyman I. Goldstein

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### STEATORRHEA AND GLOSSITIS AFTER ILEOCOLOSTOMY—EFFECT OF SYNTHETIC VITAMINS OF B-COMPLEX, AUTOLYSED YEAST, AND LIVER EXTRACT. A. Brown. Brit. M. J. 1073-1076, (June 18), 1949.

A case is reported of a patient who, after ileocolostomy for probable regional ileitis, developed steatorrhea, persistent glossitis and stomatitis. The mouth findings were controlled on several occasions by calcium pantothenate, inositol, by yeast and refined liver extract all of which were given either by mouth or intramuscular injections. The calcium pantothenate and also the liver extract when given parenterally caused abdominal pain. However, when the liver extract, yeast extract and inositol were given by mouth they cured the glossitis and did not produce abdominal discomfort. The calcium pantothenate did not improve fat or glucose absorption.

A. X. Rossien

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### DIVERTICULITIS OF THE SIGMOID—A PERSONAL EXPERIENCE. Francis Marion Pottenger. Tr. Am. Thera. Soc. 30:74-86, 1931.

In this article, Dr. Pottenger describes his own case and then discusses expertly the "toxic syndrome of diverticulitis", the "reflex symptoms of diverticulitis", the response on the part of the stomach, the response in other parts of the gastrointestinal tract, sensory reflexes, reflexes from the stomach to the diverticulum, the cause of recurrent pain (sensory phenomena); reflex in the heart. At the time of this original study (1927-1931), the author was a young man of sixty. In this unusual report, Dr. Pottenger attempts to explain in terms of vegetative neurology the major symptoms personally experienced in recurrent attacks of diverticulitis. He attempts to show by this analysis how the vegetative nervous system integrates and correlates action in different parts of the body, and how the normally submerged reflexes which are present, but not in the field of recognition in conditions of health, may disturb function and produce symptoms when they are exaggerated by the inflammatory process of disease. The various reflexes and referred sensory changes manifested during illness—are ascribed to the afferent and efferent neurons which were responsible for their production. He discusses the method by which a chronically inflamed viscus may produce recurrent pain when no active inflammation is present.

Hyman I. Goldstein

## LIVER AND BILIARY TRACT

**THE TREATMENT OF ACUTE CHOLECYSTITIS. C. G. Thomas, Jr. and N. A. Womack. S. Clin. North America. 29:1445-1455, (Oct.), 1949.**

The authors present a rational approach to the treatment of acute cholecystitis based on experimental and clinical observations.

They deal with "acute calculous cholecystitis". They do not discuss noncalculous cholecystic disease.

They advocate immediate surgery in cases of acute calculous cholecystitis when the patient is seen within a few hours after the onset of symptoms. Deferment of operation with close observations of the patient is the procedure of choice where there has been some delay, because of the high incidence of focal gangrene and the tendency toward restitution. Then, these delayed cases can be more successfully treated surgically several weeks after complete subsidence of symptoms.

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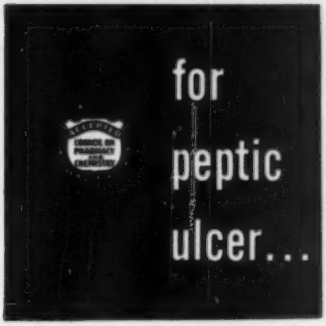


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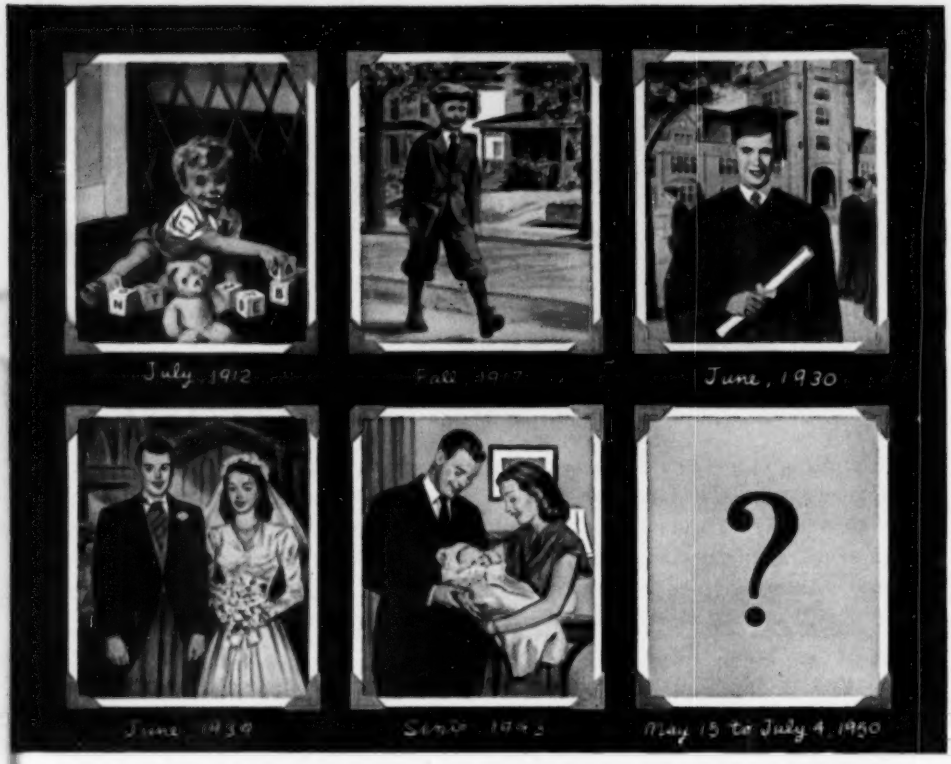


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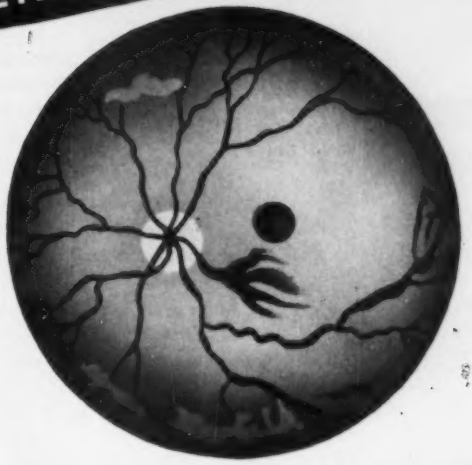

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**Hulsher, O., Lancet,* ***251****, 272 (1946).*

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*Rivers, A. D., Am. J. Dig. Dis., **2**, 189 (1935).

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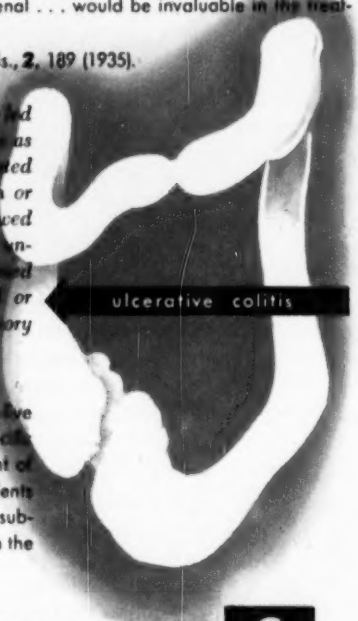
**Gill, A. M., Lancet,* ***2****, (1945).*

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*Streicher, M. H., J. Lab. Clin. Med., **33**, 1633 (1948).

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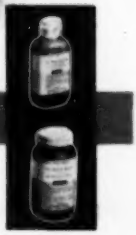

Literature available upon request

**Viodenum**

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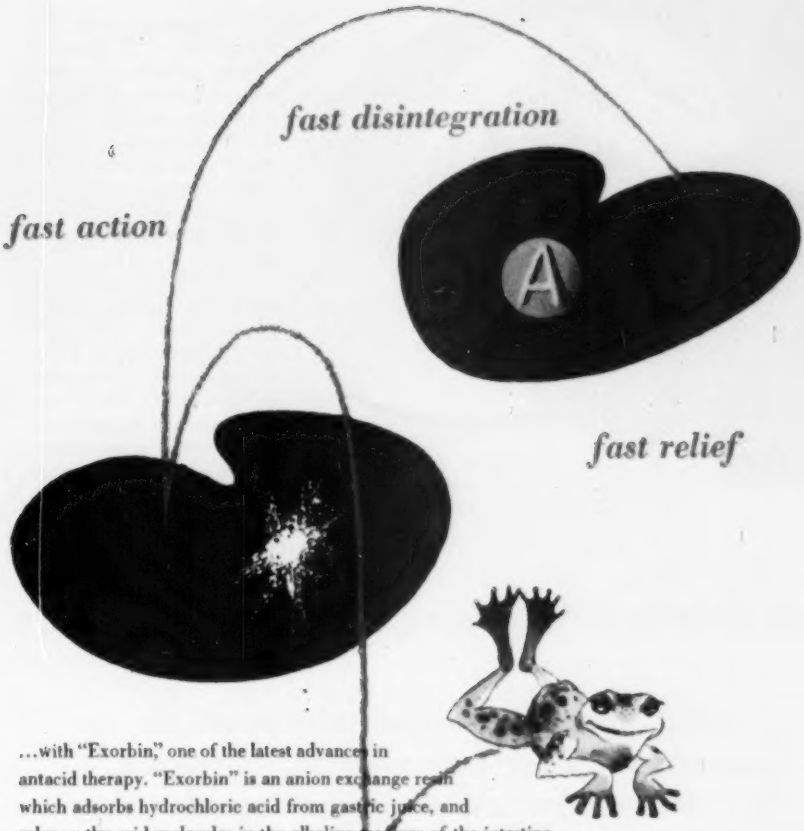


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[1] Kraemer, M.: Postgrad. Med. 2:431 (Dec.) 1947.
[2] Kraemer, M., and Siegel, L. H.: Arch. Surg. 56:318 (Mar.) 1948.
[3] Martin, G. J., and Wilkinson, J.: Gastroenterology 6:315 (Apr.) 1946.

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BIBLIOGRAPHY
1. WILLIAMS, V. P.: New England J. Med *236*: 322, 1947.
2. KARNOSH, L. J. and ZUCKER, E. N.: A Handbook of Psychiatry, St. Louis, Mosby, 1945.

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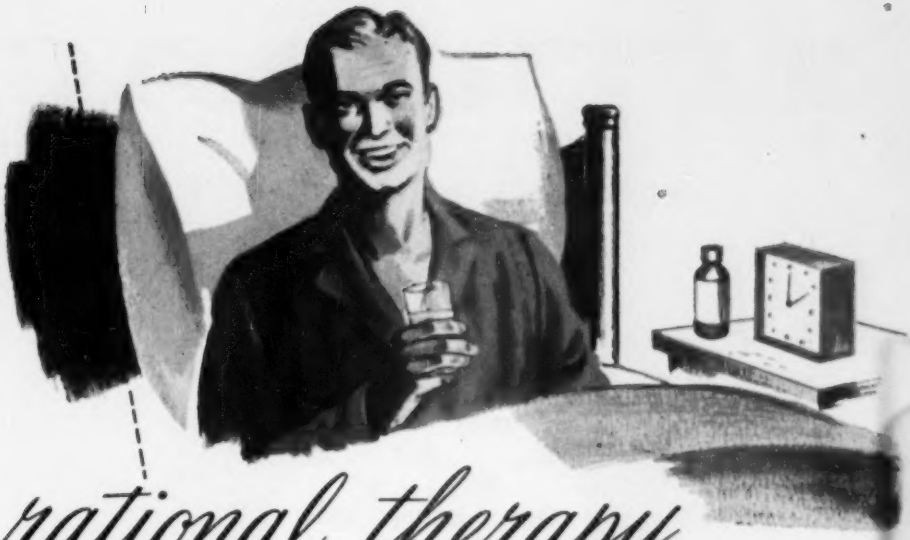

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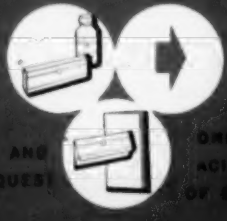

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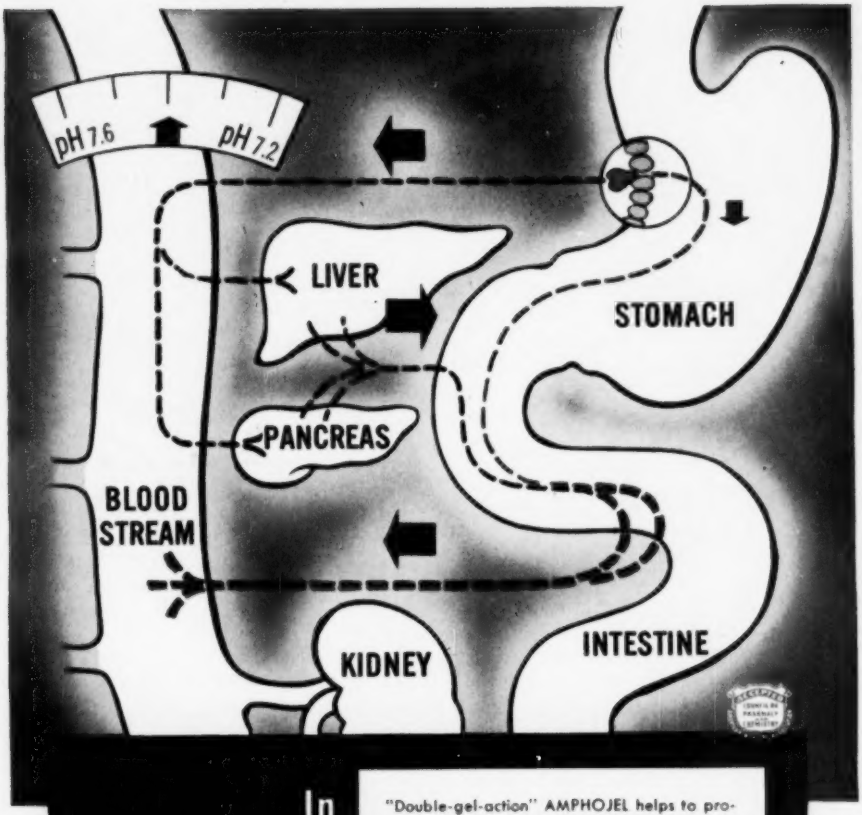


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